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JULY, 1943.

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Royal College of Surgeons of England.

JACKSONIAN PRIZE.

THE Council of the Royal Australasian College of Surgeons has been advised by the Council of the Royal College of Surgeons of England that the subject chosen for the Jacksonian Prize for the year 1944 is as follows: "The Causation and Treatment of Delayed Union of Fractures of the Long Bones." Full details governing the conditions of the prize will be announced at a later date.

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Authors are requested to submit two typescript copies of all articles.

References to articles and books should be carefully checked. In a reference the following information should be given without any abbreviation: initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given, with full date in each instance.

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THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY

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THE TREATMENT OF BONE CAVITIES.[1]

By BALCOMBE QUICK,
Melbourne.

THE obliteration of any abscess cavity is normally brought about by the approximation of its bounding walls and the gradual coalescence of the granulation tissue covering them, the so-called healing by third intention. In the soft parts this approximation is brought about largely by the maturation of fibroblasts into fibrous tissue, aided at times, no doubt, by the tonus and tension of neighbouring tissues, muscular or otherwise. There are, however, a number of chronic suppurative conditions, quite analogous to chronic abscess cavity, in which any healing process of this kind is rendered difficult or impossible by the rigidity of the bounding walls. Instances of this kind spring to mind at once—chronic empyema and bronchiectatic cavities, suppurating calcified hydatid cyst, as well as chronic osteomyelitic or any other cavity formation in bone associated with infection.

The difficulty attending the treatment of any of these conditions is well known to every surgeon, and in all the common factor in difficulty is the rigid wall. In the case of bone the problem is further complicated by the fact that this same rigidity is essential to function, and no operation comparable to, let us say, thoracoplasty is practicable. The problem is a difficult one and one which, when it occurs in such situations as the trigone of the femur or the interior of the *os calcis*, has been looked upon by many surgeons as insoluble by conservative methods. It is a problem which began to assume a considerable importance twenty-five years ago and which seems likely to loom large again in the near future. It occurs in relation both to the ordinary hæmatogenous osteomyelitis of early life and to that following infection of a compound fracture, and particularly those infected compound fractures caused by the missiles of warfare.

The history usually given by the possessor of a chronic bone cavity is one of repeated operations, in the earlier of which, at any rate, sequestra have been removed. Healing may even have been secured for a time, only to be followed sooner or later by a recurrence of pain and fever, relieved after incision or a return of discharge from the old sinuses.

Examination will now often reveal a cavity of greater or lesser extent whose walls are formed of bone showing radiologically a varying degree of sclerosis and covered by a more or less vascular granulation tissue. The wall may be formed in large part of bone originally laid down as involucrum or callus, or maybe the dense cortical bone of the original shaft (Figure I).

[1] Accepted for publication on December 4, 1942.

In the latter event the medulla or cancellous tissue has long since disappeared, either by a process of caries or as a result of curettage. In either event it is probable that in former sequestrectomies or "scrapings" there has been some degree of guttering or unroofing of the cavity for the necessary access (Figure II).

It is notable that it is towards the ends of the long bones, in the vicinity of the joints, where the anatomy is becoming more crowded and a little more

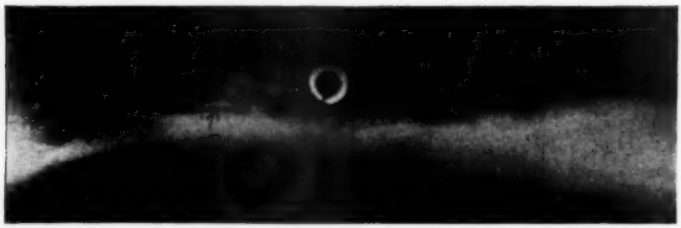


FIGURE I. Bone cavitation with sequestra and considerable surrounding sclerosis. A marker indicates the position of a sinus.

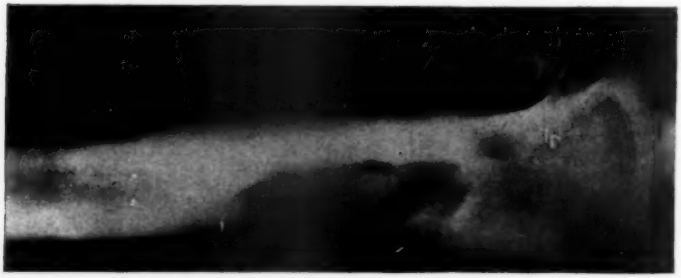


FIGURE II. Partially unroofed cavity, with bone sclerosis.

FIGURE III. A cavity with "B.I.P.P." pack *in situ*.

complex, and muscle bellies are giving place to tendons, that the greatest difficulties are met. Here it is not uncommon to find that the cavity not only extends into the remains of the medullary canal in either direction, but that the joint-wise extension may involve the cancellous bone of the articular enlargement itself. Towards the middle of the shaft conditions are less difficult in every way and long-standing bone suppurations are correspondingly less frequent.

The methods that have been employed in the treatment of such cavities involve two different principles. In both the cavity is unroofed, planified, flattened out, or, as the process has been termed, "saucerized" to some degree. This preliminary step is an essential one. In it the absence of sequestra is verified and search made for any sinuses or tracks leading through the bone in the depths of the cavity into the soft tissues beyond. This preliminary completed—and it involves a suitably extensive incision and full exposure of the involved part of the bone — the remainder of the operation will be determined by the choice of method then made.

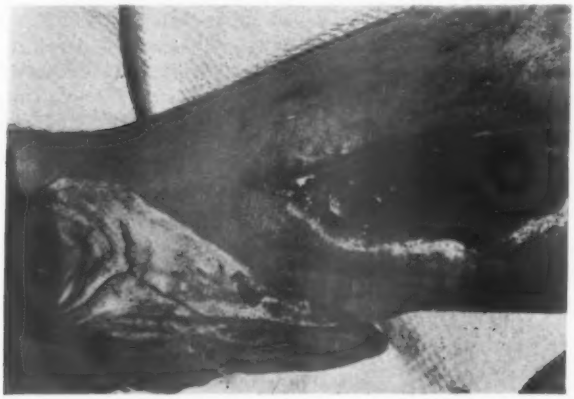


FIGURE IV. Cavity after removal of the pack. The inlay has been prepared.

METHOD I.

In the first method the cavity is filled and obliterated by the introduction of: (*a*) an antiseptic paste or wax, such as that of Mosetig-Moorhoff; (*b*) fat grafts, either free or pedicled, but usually the former; (*c*) a pedicled muscle graft, retaining its own blood supply.

Of the paste and free fat grafts personal experience has been very limited, for the reason that a short trial appeared to confirm the grave theoretical defects inherent in these methods. A study of Rutherford Morison's results fails to carry conviction that his free fat grafts really survive, and it is probable that both these procedures are futile. For the use of a vascular pedicled graft of muscle there is a good deal to be said. Various surgeons have reported series of cases treated in this way

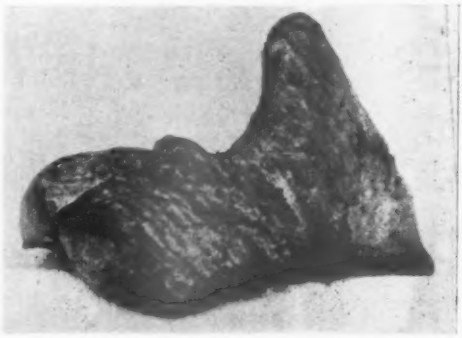


FIGURE V. Thiersch grafts applied to inlay.

FIGURE VI. An inlay used in cavity of lower end of humerus following compound fracture.

with considerable success. The writer's own small experience has not been uniformly satisfactory. The difficulty of avoiding such kinking or angulation of the pedicle as must jeopardize the blood supply is very real, and there are certain situations, moreover, in which a suitable graft is unobtainable, such as the outer side of the head of the tibia and the whole region of the ankle joint. It is always likely too that widespread scarring of the adjacent skin may make its closure impossible at the end of the operation, however well the muscle graft may have been adjusted to the cavity. A skin plastic of some kind may therefore be required to complete healing.

METHOD II.

In the second method the obliteration of the cavity is not sought. The cavity is accepted and the sole aim of treatment is to secure healing by providing a covering of epithelium to it. This epithelium has usually been secured by turning into the cavity one or more pedicled flaps of skin and subcutaneous fat. The latter is, of course, essential to the nutrition of the skin, which without it would inevitably slough. The cutting of skin flaps in the vicinity of much old scarring demands a great nicety in judgement. It is likely that the only practical solution in such a case may be found in bringing a pedicled flap from a distance. There are other definite technical difficulties connected with the use of pedicled flaps of skin and its underlying fat. Their bulk renders introduction into a cavity, even one which has been quite extensively unroofed, rather difficult, particularly if two such flaps are employed. Difficulty may be experienced too in securing fixation *in situ* during the healing period. Enough has been said to draw attention to the real difficulties that are liable to be encountered in the pursuit of this method of treatment.

FIGURE VII.

The method to be described here has been used in a considerable number of cases during the last twenty years. Like that just described, it is a non-obliterative procedure, but in place of a skin flap to cover the granulations on the walls of the cavity epithelialization is secured with free Thiersch grafts. The introduction of the grafts and their secure retention and immobilization are carried out after the well-known method of the Esser epithelial inlay.

The first occasion on which this plan of treatment was used was in dealing with a sinus leading completely through the head of the tibia. Success attended the application of grafts upon conical moulds, introduced from both sides and meeting at their apices in the centre of the tibial head. Not long afterwards a cavity in the *os calcis* was induced to heal, leaving sufficient parietal bone for full weight-bearing and activity.

The method finds its most useful field in the case of cavities near the ends of the long bones. For here, as pointed out earlier, almost insuperable difficulties may be met in the trial of any other method. Since the epithelial

covering is well "recessed", that common defect of a Thiersch-grafted surface—inability to stand up to wear and tear—is not encountered.

Preparation of the Cavity.

The preparation of the cavity is important, for without a suitable surface upon which to place the grafts success will not be obtained. In the first place it is necessary that the shape of the cavity should be such that the introduction and removal of the mould of dental composition are practicable. This necessitates a smooth walled cavity which tapers gradually throughout its extent, from the surface to its furthest limit. It may be noted that this does not imply the complete unroofing of the cavity, much less its "saucerization", by any means (Figure III). This shaping of the cavity calls for the use of chisel and gouge. During this stage it is well to bear in mind that the denser and more sclerotic the bone is found to be, the wider should be the removal.

FIGURE VIII.

Following the appropriate shaping of the cavity to receive an inlay, both the cavity itself and the approach through the overlying soft parts are firmly packed with "B.I.P.P." gauze (Figure III). This packing is not disturbed for ten to fourteen days, or even longer, the period depending upon the estimated degree of sclerosis of the walls. Bone which is sclerotic and comparatively avascular is unsuitable for the immediate reception of grafts, and infected bone is obviously hopeless in this respect. Recently sectioned bone, even though living and

FIGURE IX.

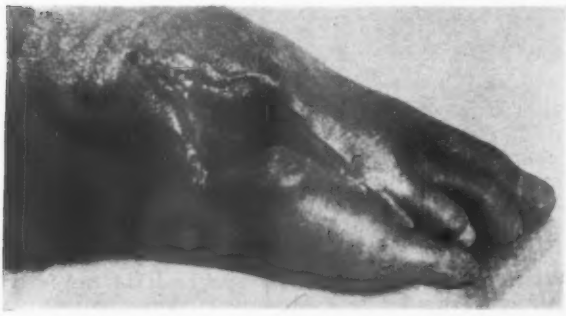


FIGURE X.

vascular, is of very doubtful value in the presence of infection. The ideal surface is one in which infection has been reduced to a minimum and where the bone has become covered with healthy granulation tissue, florid and vascular, not sodden and œdematous.

To secure such a surface demands a period of preparation and delay following the exploration and regularization of the cavity. The length of this period, during which granulations spring from the surface of the bone, is largely determined by the chronicity of the condition and the consequent sclerosis of the bone. The less vascular the bone at the sites of section, the greater the necessary delay. Although in many cases the walls are found covered with suitable granulation tissue in ten to fourteen days after

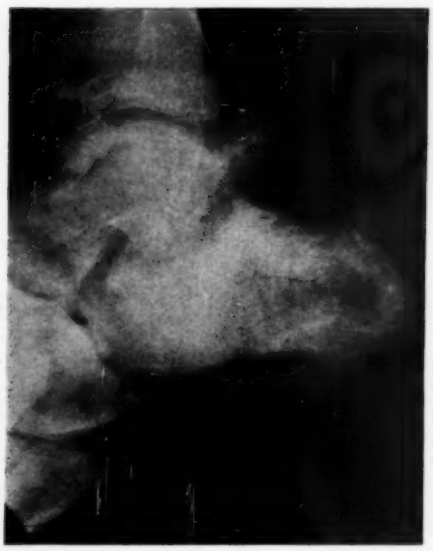


FIGURE XI. Chronic osteomyelitis of *os calcis*, with involvement of astragalo-calcaneal articulation and part of astragalus. Multiple sinuses.

FIGURE XII. Cavity epithelialized and soundly healed a month later.

regularization of the cavity and packing, in others many weeks may elapse before conditions are favourable for the application of grafts, for the complete removal of sclerotic bone may have been impracticable.

Preparation of the Mould and of Grafts.

If, upon removal of the packing, suitable granulations are still lacking, the whole area is cleaned up and repacked until conditions are finally found to be satisfactory. When this is so the cavity is irrigated with Dakin's solution and the mould prepared, the dental composition being heated until thoroughly plastic and well kneaded into the cavity to be grafted (Figure IV). The introduction of a Watson-Cheyne dissector into the outer part of the mould at this stage furnishes a handle which is of the greatest convenience during the subsequent steps. Following the cooling and setting of the mould—which may be hastened by the application of an ethyl chloride spray—it is carefully withdrawn by the dissector and plunged into cold water. When quite firm it is well to determine by reintroduction that deformation of the mould has

not occurred and that the fit is accurate throughout. The whole of the surface which is in contact with granulation tissue is then covered with really thin Thiersch grafts, which are best cut in sufficient length to extend right across the mould, from base to apex and back to the opposite side of the base. To secure the initial adhesion the old plan of smearing the surface of the mould with blood has been found satisfactory and the use of "Mastisol" has not appeared necessary. The whole surface should be covered without overlapping of the edges of the grafts (Figure V). The raw surface of the grafts, needless to say, is outermost.

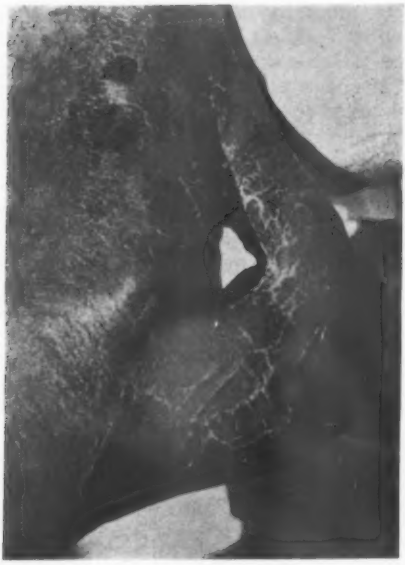


FIGURE XIII. The bilateral approach is shown. The *tendo Achillis* functions, despite its isolation.

Introduction of the Dressed Mould and After-Treatment.

It is at this point, when the introduction of the dressed mould might be attempted forthwith, that displacement of the grafts would be almost certain to occur. The problem is very simply solved by freezing—again with ethyl chloride spray—thus converting the filmy, slippery grafts into rigid and immobile components of the mould. A few minutes spent in carefully spraying a fine jet of ethyl chloride (as marketed for local anæsthesia) over the whole surface overcomes all difficulties of introduction, converting an irritating and rather futile attempt into a certainly successful procedure.

When the mould has been thus slipped home into its cavity it is surrounded and covered with a suitable gauze dressing. A rubber sponge is then adjusted over all with strapping, so that an adequate degree of pressure is maintained throughout. In many situations the application of plaster of Paris may be desirable to secure the necessary complete immobility. The mould is left untouched until its removal about the seventh day, or a little later if the surrounding gauze shows no sign of discharge (Figures VI and VII). The presence of some discharge will indicate that the take has not been universal; but, even so, epithelialization is generally quite complete within two or three weeks. The cavity meanwhile is kept rather firmly packed with "Vaseline" gauze, which is changed every two or three

FIGURE XIV. Patchy areas of bone sclerosis in the floor of an otherwise healed cavity.

The extreme cold has no devitalizing effect whatever upon the grafts.

days. The dependent position should be avoided for about a month from the time of application of the grafts. Neglect of this precaution may result, in spite of firm support by packing, in subepithelial ecchymosis from rupture of newly formed capillaries. It is necessary to warn the owner that care must be taken of the cavity even after healing is complete. It must be kept free of epithelial debris, for the surface desquamates rather freely, and of fluff from woollen underwear, and it must be dried out after the bath. A dusting with boracic acid and a very light packing of absorbent wool are a small price to pay for a healed cavity (Figures VIII, IX, X, XI, XII, XIII). The after-history of the healed bone cavity is interesting. Although actual measurements in proof are lacking it appears quite certain (an opinion shared with sundry patients) that in the course of months and years it has a tendency to diminish in size.

It would be idle to claim that all cases treated in the way described go on at once to a complete and final healing. From time to time it will be found that bone sclerosis is of such a grade that the formation of suitable granulations upon which to place the grafts fails to become general. In such event a patchy failure to take becomes evident. In the course of time sclerotic flakes (Figure XIV) may separate or be removed, whereupon the area exposed in this way may still epithelialize. It may be impracticable to carry out the primary preparation of the cavity as thoroughly as desirable, on account of possible serious weakening if all sclerotic bone is removed. This risk will be particularly evident when the floor of the cavity, directly opposite the site of the unroofing, has been found to be affected in this way. Even with subtotal healing any residual discharge is minimal and easily cared for with a very small dressing, often worn in the cavity itself.

In conclusion, it may be added that there is apparently an increased immunity from serious recurrences of deep bone infection. In the event of the removal of infected bone at the preparatory operation having been incomplete, there may be a recrudescence of activity in the organisms months or years later. This is a risk from which no bone infection is ever free. But under the plan of treatment which has been described the organisms are not buried in the depths of a great thickness of scar tissue. The focus of activity is but fractionally beneath the epithelial covering, and thus tension is relieved almost before constitutional signs become evident.

CEREBRAL ŒDEMA.[1]

By W. Lister Reid,

Consulting Neurosurgeon to an Australian General Hospital; Honorary Neurosurgeon, Lewisham Hospital, Sydney; Honorary Assistant Neurosurgeon, Royal Prince Alfred Hospital, Sydney.

(From the Department of Surgery, University of Sydney. Aided by a grant from the National Health and Medical Research Council.)

Ætiology and Diagnosis.

Cerebral œdema has been recognized during recent years as a clinical entity of paramount importance in the realms of neurosurgery. Modern methods of investigation have enabled us to diagnose this condition at an early stage, and improvements in histological technique have afforded greater facilities for the study of its histopathology. With this knowledge it is possible to recognize œdema as a complicating factor in many intracranial and extracranial pathological lesions; and it has been shown to resemble œdema elsewhere in the body, in that it is due to an excess of intracellular and interstitial fluid. When material is available for microscopic study, varying degrees of swelling of the cellular and supporting structures of the brain may be seen, as will be shown in the following pages.

When tissue is not procurable for examination, the presence of œdema is largely assumptive and based on the clinical evidence, and on the histological appearance of tissues obtained from similar cases. In my experience, varying degrees of œdema were invariably found in acute inflammatory lesions and in intracranial expanding lesions, providing these latter were present for more than a few hours.

Before the causes of cerebral œdema are considered in detail, it will be well to discuss its symptomatology in brief. The symptoms and signs depend largely on whether the œdema is generalized or confined to a small area. The following are the main clinical features that may be presented as the result of generalized œdema:

1. A history of cerebral trauma; a history of intoxication by some agent such as lead; or the presence of an intracranial inflammatory or expanding lesion.
2. A latent period of several hours between the cause, such as trauma *et cetera*, and the onset of symptoms.
3. Headache, which is usually generalized.
4. Drowsiness, restlessness and irritability.
5. Vomiting.
6. Double vision, due to ocular nerve palsies, especially the sixth nerve.
7. Epileptic seizures.
8. Elevation of the intracranial pressure and an increased secretion of cerebro-spinal fluid.
9. Recovery almost invariably occurs when the causative lesion is removed, and the recovery is usually greatly hastened by the intravenous administration of hypertonic solutions.

[1] Accepted for publication on April 22, 1942.

When the œdema is localized to a relatively small area, the symptoms and signs are correspondingly focal, depending on the site and extent of the œdema. The signs presented are usually unilateral, and may consist of ocular nerve palsies, focal Jacksonian seizures, aphasia and motor disturbances varying from localized weakness to hemiplegia. It should be noted that the symptoms and signs enumerated above correspond closely with those produced by any intracranial expanding lesion. This is probably due partly to the œdematous tissue acting as an expanding lesion, and partly to tissue anoxæmia produced by the excess fluid in and around the cells and by interference with the intracerebral blood flow. I wish to draw particular attention to this, and to emphasize that cerebral œdema should never be diagnosed until it is absolutely certain that the patient's condition is not due to an expanding lesion such as a neoplasm, cyst, abscess *et cetera*.

The commonest causes of cerebral œdema may be classified under three headings: traumatic, inflammatory and toxic.

Traumatic Œdema.

The usual causes of trauma to the brain are: (*a*) a blow on the head, (*b*) operative procedures on the brain, (*c*) electrical stimulation of the cerebral cortex, (*d*) pressure exerted on the surrounding brain tissue by an intracranial expanding lesion. In those cases in which death occurs instantaneously following a head injury, there is no evidence of cerebral œdema, providing that the autopsy is performed within a short time of death. If the post-mortem examination is delayed too long, some evidence of swelling can be found in the cellular and interstitial tissues.

Head Injury.—In cases of head injury in which death does not occur, the diagnosis of the presence of œdema is largely presumptive, and must be based mainly on the examiner's knowledge of the microscopic findings in tissues obtained from similar cases at operation or autopsy. I have found the following points useful in recognizing the presence of œdema: (i) The latent period of several hours between the trauma and the onset of symptoms. (ii) The increased intracranial pressure, the increased production of cerebro-spinal fluid and the absence of blood in the cerebro-spinal fluid. (iii) The rapid amelioration of the symptoms and signs following the intravenous administration of hypertonic glucose or sucrose solutions. These factors are by no means pathognomonic of the presence of cerebral œdema, as they may also occur as the result of a lesion such as an extradural hæmorrhage, and even be partially relieved by intravenous hypertonic solutions.

Focal signs such as paralysis, Jacksonian seizures, aphasia *et cetera*, following a head injury, should be regarded with concern, as they may be due to cerebral laceration, to pressure by a depressed piece of bone or to a subdural, extradural or intracerebral hæmorrhage. The absence of blood in the cerebro-spinal fluid is usually sufficient to exclude any laceration of the brain, except in cases in which the laceration is deep in the *centrum ovale* and unconnected with the subarachnoid spaces. Extradural hæmorrhage is generally fairly easy to detect, and should be suspected if, after a latent period of some hours, the patient becomes increasingly drowsy and exhibits signs of pyramidal involvement, ipsilateral third nerve palsy or mid-brain compression. Further evidence may be supplied when X-ray examination reveals a fracture line crossing the path of the middle meningeal artery or one of its branches.

Subdural hæmorrhage is much more difficult to distinguish from cerebral œdema, as it rarely presents any localizing signs. In most cases it is merely suggested by the lack of improvement in the patient's condition and by persistent headaches, slightly elevated cerebro-spinal fluid pressure, slight increase in the cerebro-spinal fluid protein content, and increasing drowsiness

in the later stages. The discovery of a fracture may aid the diagnosis, and as a rule the condition occurs in elderly people in whom the dura is more firmly attached to the overlying bone than in youth. The symptoms are slow in their onset and often extend over a period of weeks.

Intracerebral hæmorrhage may also be very difficult to distinguish from cerebral œdema. It acts as an intracerebral expanding lesion and causes signs depending on its situation. It may or may not be associated with blood in the cerebro-spinal fluid. Unless its position can be localized clinically, the surgeon may have to resort to exploratory puncture or encephalography.

Operative Trauma.—Operative trauma may be produced by too forceful retraction of the brain or too violent pressure or traction during the removal of an intracerebral expanding lesion. Occasionally abnormal signs make their appearance some hours after such an operation, and it is essential that the surgeon should decide whether they are due to cerebral œdema or to post-operative hæmorrhage or thrombosis. If the signs are due to œdema they usually occur from about six to twenty-four hours after the operation. They are generally focal in nature, and consist of motor or sensory disturbances, aphasia, Jacksonian seizures and double vision. The general condition of the patient is not greatly affected as a rule. The main point in favour of the presence of œdema is the good response to the intravenous administration of hypertonic solutions, and the spontaneous recovery after a few days if left untreated.

Post-operative hæmorrhage is more rapid in its onset and, if unrelieved, produces a steady progression of signs which may end in coma and death. There is little or no improvement following the injection of hypertonic solutions. Post-operative thrombosis is also fairly slow in its onset, but there is no improvement with hypertonic solutions. Spontaneous recovery may occur, but it takes place over a period of weeks. It is often extremely difficult to distinguish between these three conditions, especially as hæmorrhage and thrombosis are almost invariably accompanied by a certain degree of œdema. More often than not, the final opinion depends on the previous experience of the surgeon rather than on any definite set of rules.

Electrical Stimulation of the Cerebral Cortex.—Electrical stimulation of the cerebral cortex, if prolonged and of high intensity, is another form of operative trauma. Following the performance of this procedure on epileptic patients, even when no brain tissue is removed, one not infrequently sees a series of epileptic seizures twenty-four hours or so after the operation. I have not seen this occur in non-epileptic patients. The seizures abate of their own accord within a day or so, but they usually stop immediately following the intravenous administration of hypertonic glucose or sucrose solution. This strongly suggests that cerebral œdema plays a large part in their production, occurring particularly in an epileptogenic brain. Further evidence for this view may be presented by the fact that, in epileptic patients, seizures can be induced by increasing the intracerebral fluid content. This is utilized in the clinical investigation of the epileptic. The patient is hydrated by forcing the fluid intake and by reducing the fluid output by injections of pitressin. Seizures usually follow this procedure and the examiner has the opportunity of personal observation of the pattern of the attack. The work of Pupo[1] also supports this view. By experimenting on rabbits, he showed that the convulsive threshold for "Metrazol" was lowered by the presence of cerebral œdema and greatly raised by dehydration of the brain. He found that by injecting distilled water into rabbits the dose of "Metrazol" necessary to induce convulsions dropped from 0·2 or 0·3 cubic centimetre to 0·1 cubic centimetre. When a 50% solution of dextrose was injected the amount of

"Metrazol" required to produce a convulsion was increased to 1·6 cubic centimetres.

Intracerebral Expanding Lesion.—It is extremely difficult and frequently impossible to distinguish between the symptoms produced by an intracerebral expanding lesion and those due to the accompanying œdema. Varying degrees of œdema were found in all the brains that I examined, which comprised those affected by neoplasm, hæmorrhage, cyst and abscess, providing that the lesion had been present for at least some hours. It must be realized that œdema plays only a small part in the production of symptoms in these cases, the main factors being the pressure exerted by the expansion of the lesion and the destruction of the surrounding tissue. Exceptions to this rule are metastatic deposits and certain cases of small abscess formation. In some of these cases the symptoms and signs are out of all proportion to the size of the lesion. This is due to the marked and widespread distribution of the accompanying œdema.

In order to emphasize some of the points described above, and to illustrate the difficulties that may beset the neurosurgeon, I should like to present the case of a patient who recently came under my care at Lewisham Hospital.

E.N., a male patient, aged forty-eight years, was admitted to hospital by ambulance. Some thirty hours prior to admission he received an injury to the right side of his head in a drunken brawl, but it was not known whether he was rendered unconscious. On arrival at his home six hours later he appeared dazed and had some cuts on his face; but examination revealed no other abnormality. He went to bed and slept for several hours, and on awakening he was rather restless and complained of some headache, which was partially relieved by aspirin, and later he went to bed again.

On the following morning, about twenty-four hours after the injury, he was very difficult to rouse, and he was reported to have had focal seizures involving the left side of his face and the left arm. During the latter part of the morning the drowsiness increased, and the seizures occurred more frequently. He was brought to hospital and on admission he was in deep coma and was having focal seizures every four to five minutes, involving the left side of the face, the left arm and, to a lesser extent, the left leg. On examination there was weakness of the left side of the face, and the left arm and leg did not move voluntarily. There was nothing of localizing value in the deep reflexes, which were generally diminished, but the left abdominal reflex was absent and there was an extensor plantar response of the left big toe. Lumbar puncture revealed a cerebro-spinal fluid pressure of 240 millimetres of water and the fluid was blood-stained, but an X-ray examination of the skull revealed no evidence of a fracture.

Because of the delay of twenty hours or more before the onset of the seizures, it was felt that they were probably due to the presence of cerebral œdema. The hemiplegia was assumed to be due partly to œdema and partly to cerebral laceration, as evidenced by the presence of blood in the cerebro-spinal fluid. The coma was believed to be due to increased intracranial pressure produced by the œdema and increased cerebro-spinal fluid secretion. On this basis of thought the patient was given 200 cubic centimetres of 25% glucose solution intravenously over a period of forty minutes (sucrose solution was not available).

During the period of injection and during the next hour or so the seizures gradually decreased in frequency and severity, until they ceased altogether; and they did not recur during the remainder of the patient's stay in hospital. He slowly recovered consciousness during the following six days, and power partly returned to the left side. On the sixth morning, however, he became rather drowsy and difficult to rouse, and during the course of the day he again lapsed into deep coma with signs of complete left-sided hemiplegia. It was felt that this change in his condition was due either to a delayed intracerebral hæmorrhage or to a subdural hæmatoma.

The patient was taken to the operating theatre and a small burr hole was made in the right temporal region under local anæsthesia. A fairly large subdural hæmatoma was found pushing the brain about two centimetres away from the bone, and this was thoroughly washed out with Ringer's solution through the burr hole. The wound was then closed and a small rubber drain left beneath the bone through a separate stab wound for twenty-four hours. On the following day the patient's condition was much improved. He was quite conscious, although still very drowsy, and there was some return of movement to the left side of his face and left leg. During the remainder of his stay in hospital his condition continued to improve slowly until a full range

of movement returned to the face and leg, and partial movement to the arm. No further seizures occurred. On his discharge from hospital recovery was complete, except for slight spasticity of the left arm with some limitation of movement.

Inflammatory Œdema.

Microscopic evidence of œdema can always be found in cases of intracranial inflammatory lesions, the degree of œdema depending on the extent and acuteness of the inflammation. In cases of meningitis swelling of the cells can be seen throughout the whole brain, even in structures that are most distal to the actual site of inflammatory reaction. In cases of abscess formation the œdema is usually confined to the neighbourhood of the abscess while it is relatively small, but becomes more generalized as the abscess increases in size. This generalized œdema is probably due to partial obstruction of the cerebral venous drainage, as the result of increased intracranial pressure, rather than to actual inflammatory reaction. The microscopic changes found in the tissues around an abscess cavity, and in the subpial tissues at the base of the brain in meningitis, are much more marked than in simple post-traumatic œdema. It would seem that something more than pure œdema is necessary to account for the extreme cellular swelling and destruction that are found around such lesions.

The diagnosis of the presence of œdema in these cases is based on histological evidence, as it is invariably found on microscopic examination of tissue obtained at operation or autopsy. Occasionally one sees cases in which the intracranial pressure is very high, but at operation or autopsy the abscess is found to be relatively small. In these cases there is always marked generalized œdema throughout the brain, and this is probably the main cause of the increased intracranial tension. It is usually very difficult to assess the exact size of an acute abscess without pneumographic visualization of the ventricles, because the surrounding œdema plays quite a large part in the production of symptoms.

Toxic Œdema.

In the preceding group cerebral œdema has been discussed in relation to intracerebral inflammatory lesions. There is another closely allied group in which the œdema occurs as a distinct entity in the absence of any intracerebral lesion. It is not an uncommon condition and it is usually associated with some form of extracranial focal sepsis, such as aural, dental, sinus, respiratory sepsis *et cetera*. Sometimes it occurs in certain types of intoxication, particularly with lead. Middle ear disease is the most commonly associated lesion, and in such cases the intracerebral condition is frequently referred to as "otitic hydrocephalus". When it occurs as the result of extracranial inflammatory lesions, such as dental, sinus or respiratory infections *et cetera*, it is often spoken of as "toxic hydrocephalus". These patients usually recover, and I have not had the opportunity of studying the tissue microscopically, so that as far as this group is concerned the presence of œdema is largely assumptive and based on the clinical findings.

The recognition of œdema as the cause of the symptoms in these circumstances is very difficult owing to its close simulation of an intracranial expanding lesion. The diagnosis is generally made only by a ventricular tap or by ventriculographic air studies. The main points on which a diagnosis of cerebral œdema of toxic origin is made, are: (i) A recent or concurrent history of, or the discovery of, some form of focal sepsis in the ears, sinuses, teeth, respiratory tract *et cetera*, or of intoxication by some substance such as lead. (ii) A fairly rapid onset of headache which in severe cases may be accompanied by nausea, vomiting, drowsiness, papillœdema, weakness of one or more of the extraocular muscles, and usually a complete absence of

localizing signs. (iii) An increase in the cerebro-spinal fluid pressure which may reach as high a level as several hundred millimetres of water. (iv) Ventricular puncture. When a brain needle is inserted into the lateral ventricles there is a sudden spurt of fluid under increased pressure and then the flow, with equal suddenness, stops. This is usually indicative of small ventricles, the walls of which collapse onto the point of the needle as soon as the pressure is released. I have also experienced this phenomenon in patients with multiple small metastatic carcinomatous deposits. In such cases it is probably due to the accompanying œdema which is usually quite extensive around even small metastatic tumours. (v) Ventriculography. In the vast majority of cases it is folly to label a condition as toxic œdema without having first visualized the ventricular system by air studies. In some cases in which an extracranial focus is obvious and the condition of the patient is not extreme, it is justifiable to treat the septic focus first and see if there is any improvement. In severe cases ventriculography should be carried out in preference to encephalography, as the latter is too dangerous in cases of high intracranial pressure. In mild cases the ventricles may be found to be normal in size, shape and position. In severe cases the ventricles are compressed to mere slits, but there is no displacement to one or other side of the mid-line.

In concluding this section I would again repeat this warning. Cerebral œdema should never be diagnosed as being the sole cause of any symptoms or signs presented until all possibility of a focal expanding lesion has been excluded. This can be done only by a careful history and examination, by noting the pressure of the cerebro-spinal fluid and its biochemistry, by X-ray examination of the skull and by pneumographic visualization of the ventricular system.

REVIEW OF THE LITERATURE.

Relatively few publications have been made on the subject of cerebral œdema, when one considers the frequency with which this condition is encountered in neurosurgery. In this section summaries have been made of the findings and impressions of those who have dealt with the subject. Some have presented the changes found in the brains of man and laboratory animals following cerebral trauma, and in experimental œdema produced by the intravenous injection of distilled water into animals. Others have described the changes found in the cells and supporting structures around intracranial expanding lesions.

A few, such as Spatz,[2] Schienker[3] and de Crinis,[4] have subdivided œdema of the brain into "brain swelling" and "cerebral œdema". Spatz stated that in "brain swelling" the brain was dry and showed a general increase in consistency, while in "cerebral œdema" the brain was softer and moister than normal. Schienker's differentiation depended on the distribution of the excess fluid in the tissues. He believed that in "brain swelling" the myelin sheaths, axis cylinders and glial cells were affected, whereas in "cerebral œdema" the distension was confined to the perivascular and pericellular spaces and to the interstitial tissues. De Crinis described "swelling of the brain" as being an enlargement in volume which was not a direct result of hyperæmia, cerebral œdema or hydrocephalus. He believed that the main difference between the two was that in "brain swelling" the water content of the brain was less than in "cerebral œdema".

Most authors make no such distinction, but regard cerebral œdema as an excess of fluid in the cerebral tissues whether it be intracellular, interstitial or a combination of both. Rawling[5] was an exception, as he believed that it consisted purely of an excess of cerebro-spinal fluid. Reichardt[6] used the term "*Hirnschwellung*", which he described as a condition in which the

weight of the brain exceeded the normal figure for a given volume. He found no abnormal microscopic changes in the cases he reported. Jaburek[7] regarded cerebral œdema as an exudation of fluid into the tissue spaces, resulting in the separation of the glial meshwork and enlargement of the pericellular and perivascular spaces. Cantarrow[8] described œdema as consisting essentially of an abnormal accumulation of fluid in the interstitial compartment, and he stated that it was almost invariably dependent on some abnormality in one or more of the following factors: (*a*) the capillary blood pressure; (*b*) the colloid osmotic pressure of the blood plasma; (*c*) the relative impermeability of the capillary wall to protein, and its free permeability to water and most electrolytes; (*d*) the lymphatic circulation; (*e*) and perhaps the tissue tension or pressure resistance to the accumulation of fluid.

With the advent of the staining of nervous tissues with silver salts, Penfield and Cone[9][10] carried out an extensive survey of the neuroglia, and reported swelling of the oligodendroglia and the formation of abnormal types of astrocytes in relation to cerebral abscess, meningitis and tumour. Since this work was carried out, other authors have reported similar swelling in the glial cells, neurones and supporting structures, and have attributed these changes to œdema. Apfelbach[11] described both the macroscopic and microscopic changes in his cases of cerebral œdema. He noted on opening the skull that the dura was tight and when it was incised the brain bulged through the opening. The gyri were found to be flattened and the sulci narrowed at their peripheral ends; the veins were empty and the pial arteries almost invisible. The surface of the visceral layer of the arachnoid was dry, and by reflected light scattered, almost microscopic droplets were seen to form when the viscid serous surfaces were separated. The brain was soft and soon lost its shape when placed on a flat surface; its cut surface was light greyish-brown in colour and the lateral ventricles were closed. Microscopically the perivascular spaces were distended and empty spaces were seen between the fibres and cells, and leptomeningeal hæmorrhages were often found. The changes within the cells were usually slight and sometimes absent.

Shapiro and Jackson[12] reported the macroscopic findings in cases of death resulting from cerebral trauma. They described the traumatized brain as being dry and swollen, but not œdematous. The subarachnoid fluid was decreased, the ventricular fluid increased and there was an increase in the blood volume with the production of hæmorrhages. They assumed the presence of a certain amount of unreleasable intracellular parenchymatous fluid.

Pilcher[13] and Weed and McKibben[14] carried out studies on cerebral œdema reproduced in animals by trauma and by the intravenous injection of distilled water. Pilcher's work was carried out with regard to the fluid content of the brain following injury. He traumatized the brains of dogs by dropping weights of 500 grammes and 1,000 grammes from a distance of about five feet onto the animals' skulls. He found very little difference in the fluid content of the brain after these injuries, but there was a definite rise in the cerebro-spinal fluid pressure.

Weed and McKibben, on the other hand, studied the histological changes produced in the brains of cats following the intravenous injection of distilled water. In some animals they left the skull intact, while in others they made bilateral subtemporal decompression openings in the skull. In the former group of animals they found marked changes in the form of flattening of the gyri, narrowing of the sulci, enlargement of the spaces between the cell processes and around the blood vessels, and many of the larger dendrites were longer than usual. In the decompressed brains there were practically no abnormal findings.

With reference to the changes found around intracerebral expanding lesions, reports have been made by Spatz,[2] Schienker,[3] Greenfield[15] and others. Greenfield presents the clearest picture in this regard, and has based his report on the study of 29 cases of cerebral neoplasm and two cases of abscess. In making this report, Greenfield states that he recognizes the difference between œdema processes in relation to tumour and those in relation to general disease or intoxication. In severe cases he noted that the white matter was softer than normal and yellowish in colour, while the convolutions were wider because of the swollen *centrum ovale*. The cortex may look relatively shrunken, although actual measurements may show little, if any, change. Microscopically he noted separation and degeneration of the myelin, slight degeneration of the axones, swelling of the astrocytes in the white matter with the formation of Nissl's plump cells and an excess of fibres. The microglia were mobilized, but the oligodendroglia were relatively normal. The blood vessels were not affected and there was no widening of the Virchow-Robin spaces. The cortical neurones were sometimes degenerated in severe cases.

EXPERIMENTAL PROCEDURES.

Experimental procedures have been carried out on cats in an attempt to reproduce œdema in the brains of these animals. The object of these experiments was twofold: firstly, to present a clear histological picture of œdema as produced by an excess of fluid in the cerebral tissues; and, secondly, to confirm an impression that the changes seen around expanding lesions were due to some other factor or factors in addition to excess tissue fluid.

Seventeen cats were used in this series, of which four were used as normal controls, and each experiment was carried out under "Dial" anæsthesia. In addition to these experiments, microscopic studies have been carried out on 47 human brains to compare the picture of cerebral œdema with the changes found in the tissues around intracerebral expanding lesions. These comprised 20 cases of neoplasm, 16 cases of trauma, five cases of meningitis, three cases of extradural hæmorrhage, two cases of cerebral abscess, and one case of intracerebral hæmorrhage. In nine of the animals distilled water was injected slowly into the left common carotid artery, the arterial route being chosen to obtain the maximum hypotonic effect on the brain tissue. Of these nine animals the skull was left intact in six, while in the remaining three the calvarium and dura were completely removed to permit the brain tissue to absorb as much fluid as possible, and thus to present an extreme picture of cerebral œdema. In addition to these experiments four cats were subjected to the intraarterial injection of 25% glucose solution to demonstrate the effect of hypertonic solutions on the brain and to provide an extreme contrast to the picture of cerebral œdema.

Technique.

The apparatus used in these experiments consisted of a 500 cubic centimetre glass container fixed about fifteen feet above the level of the animal. Rubber tubing connected the container to a Murphy drip and a further length of tubing connected this with a small glass cannula. The container was then filled with distilled water or 25% glucose solution, as required, and the tubing was filled with the fluid down to the cannula tip, except for an air lock in the Murphy drip. The rate of flow was controlled by means of a screw clamp on the rubber tubing.

In each of the laboratory animals the left common carotid artery was exposed, and the tip of the cannula secured in the lumen of the vessel by means of a silk ligature. The common carotid was used because when the fluid was injected into the internal carotid the animal became distressed and soon died; and when the rate of flow was reduced to the point of safety the

blood clotted in the cannula. The rate of flow found to produce the best results was about fifty drops per minute, this being the optimum rate to prevent coagulation of the blood in the cannula and to keep the animals alive for some hours.

The experiments were divided into three groups. In Group 1, which comprised six animals, distilled water was injected into the left common carotid artery at the rate of fifty drops per minute, until the animal showed signs of expiring. The object of this procedure was to produce the greatest degree of œdema possible under normal conditions—that is, with the skull intact. In Group 2 a similar procedure was carried out on three animals, but in each case the whole of the calvarium and the underlying dura were removed. This was done to permit free expansion of the brain and consequently a greater absorption of fluid by the brain tissue. It was assumed that the resulting histological picture would represent the extreme changes possible as the result of œdema of the tissues, and that it would show the sequence of events and the type of cell *et cetera* affected by excess tissue fluid. In Group 3 a 25% glucose solution was substituted for the distilled water in four animals with their skulls intact. While this procedure had only an indirect bearing on cerebral œdema, it was done to demonstrate the changes produced by a reduction of fluid in the cerebral tissues and to furnish an extreme contrast to the picture of cerebral œdema. A fourth group consisted of four control animals. In two of these the skulls

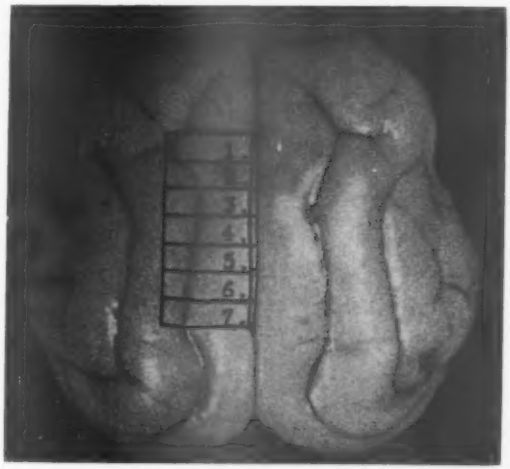


FIGURE I. Photograph of a cat's brain to demonstrate the order in which the sections were removed and placed in corresponding fixatives. (See Table I.)

TABLE I.

Section.	Fixatives.	Staining Methods.
1	Formalin 10%.	Hæmatoxylin and eosin; hæmatoxylin and Van Giesen.
2	Formalin-ammonium bromide.	Silver carbonate for oligodendroglia; silver carbonate for astrocytes; and gold chloride sublimate for astrocytes.
3	Susa's fluid.	Hæmatoxylin and eosin; and hæmatoxylin and Van Giesen.
4	Alcohol 95%.	Toluidin blue; kresyl violet; and thionin.
5	Schaudin's fluid.	Hæmatoxylin and eosin; and hæmatoxylin and Van Giesen.
6	Helley's fluid.	Hæmatoxylin and eosin; and hæmatoxylin and Van Giesen.
7	Formalin 10%.	Weigert-Pal; and Cuajunco's modification of Agduhr Bielschowsky.

were left intact, while in the remaining two the calvarium and underlying dura were removed and the brain was left exposed and kept moist with normal saline solution for six hours. This was done to eliminate any changes that might be caused by prolonged exposure to the air.

At the conclusion of each experiment, when the animal was at the point of expiring, the upper surface of the brain was photographed. This was also

done to each control animal. Seven small blocks of tissue were then removed from the left hemisphere while the animal was still alive, numbered in series from before backwards, and placed in six different fixatives (Figure I). This was done so that the sections eventually chosen for demonstration were those which showed the least distortion after fixation and staining. Each block of tissue was sectioned and stained by one or more of ten different methods. The same staining methods were used on corresponding blocks of tissue, so that accurate comparisons could be made of the cells and supporting structures of the brain. Photomicrographs were taken of the various cerebral elements to demonstrate the histological changes observed. Table I will show the manner in which the brain was sectioned, the fixatives used and the staining methods adopted.

The section fixed in Schaudin's fluid and stained with hæmatoxylin and eosin was found to be least distorted, and this tissue was used for general studies. Hæmatoxylin and eosin, and hæmatoxylin and Van Giesen stains were used to study the general architecture, the blood vessels, the ependyma, the chorioid plexus, the connective tissue and the meninges. Silver carbonate stains gave excellent pictures of the changes in the nerve cells and oligodendroglia. The Nissl bodies in the nerve cells were stained with toluidin blue, kresyl violet and thionin. Weigert-Pal's method was used to examine the myelin sheaths, and Cuajunco's modification of Agduhr Bielschowsky was used to stain the nerve fibres.

PATHOLOGY OF CEREBRAL ŒDEMA.

This section deals with the histological findings in each group of laboratory animals and in the normal controls, and the results are compared with the changes found in cerebral œdema in man. To ascertain the degree of the latter, 47 human brains were examined macroscopically and microscopically. The pathological conditions comprised 20 cases of cerebral neoplasm of various types, 16 cases of cerebral trauma, five cases of meningitis, three cases of extradural hæmorrhage, two cases of cerebral abscess, and one case of intracerebral hæmorrhage. The tissue examined for œdema was taken from areas some distance away from the site of the lesion. The appearance of the tissue in the immediate vicinity of the lesion will be discussed later.

Macroscopic Appearances.

In those animals in which distilled water was injected with the skull intact (Figure II, a), the brain bulged when the calvarium was removed, and only a small amount of clear fluid oozed out when the leptomeninges were incised. The surface of the brain was pale, the gyri widened and flattened and the sulci narrowed, while the cut surface was moist and presented a rather glazed appearance. In those animals in which the calvarium and dura had been removed prior to the commencement of the experiment, the changes were similar but more pronounced (Figure II, b). The brain was greatly swollen, the gyri enlarged but not flattened, and the sulci somewhat widened. These changes were more noticeable on the left side, on which the distilled water was injected.

The appearance of the brain, after it had been subjected to the hypertonic effect of the 25% glucose solution, provided a marked contrast to the findings described above. The whole brain was greatly reduced in size (Figure II, c), a wide space being left between the brain and the overlying dura. The gyri were shrunken and the sulci somewhat widened, while the cut surface was dry and sticky, and no fluid escaped when the leptomeninges were pierced. It is extremely doubtful whether this marked shrinkage occurs in the human brain following dehydration. I have examined the brains of patients who

have been given an intravenous injection of hypertonic sucrose solution prior to operation, and in some cases the shrinkage of the gyri has been quite apparent, although not particularly marked.

In the normal controls (Figure II, d) there was no macroscopic difference between those in which the brain was fixed immediately after its coverings were removed and those in which the brain was left exposed for some hours

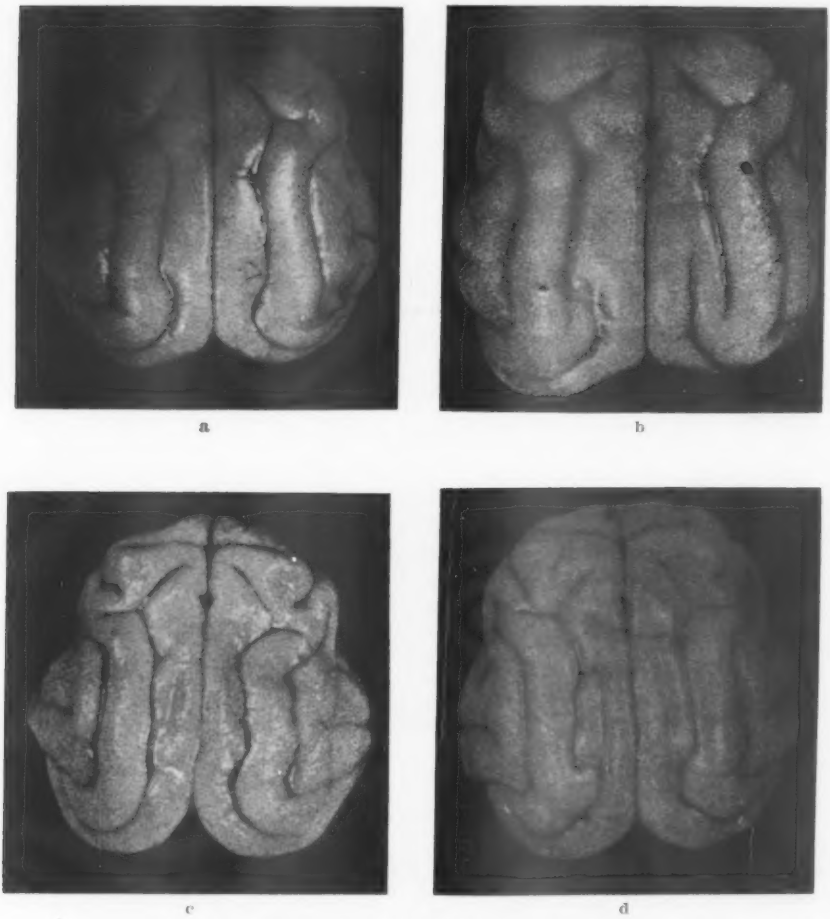


FIGURE II. Photographs showing the macroscopic appearance of the brain in the experimental animals. a: after the intraarterial injection of distilled water with the skull intact; b: after the intraarterial injection of distilled water with the calvarium and dura removed; c: after the intraarterial injection of 25% glucose solution; d: normal control.

before fixation. In human brains there were flattening and widening of the gyri and narrowing of the sulci. This was due to the increased intracerebral pressure forcing the cortex against the overlying bone. In severe cases the subarachnoid spaces appeared dry, but the cut surface of the brain was moist and had a greyish glazed appearance.

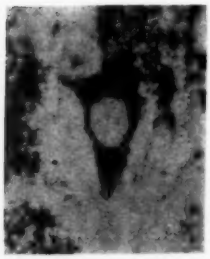


FIGURE III. Photomicrograph of a slightly swollen nerve cell. The nucleus is enlarged, ovoid and rather glazed in appearance. After distilled water with the skull intact. (Silver carbonate.)

Microscopic Appearances.

Neurones.—Little change was noted in the nerve cells after the injection of distilled water with the skull intact. In some areas the cells were larger than normal and their nuclei were somewhat distended (Figure III). Only an occasional swollen nerve cell was found in cases of cerebral œdema in man, and these were not sufficiently numerous to be regarded as a constant pathological feature.

When the calvarium and dura were removed prior to the experiment, practically all the nerve cells showed marked swelling even to the point of rupture (Figure IV). This series of illustrations shows the process of swelling in these cells, from slight distension of the cell membrane to advanced ballooning and subsequent rupture. As the intracellular fluid increased, the cell wall became distended, forming a large clear space

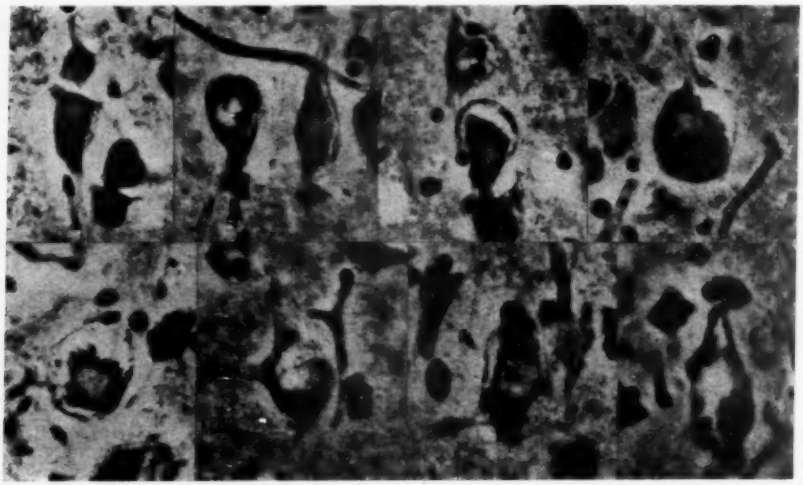


FIGURE IV. Series of photomicrographs demonstrating progressive stages in œdema of the nerve cells, from early swelling to rupture of the cell. After distilled water with the calvarium and dura removed. (Silver carbonate.)

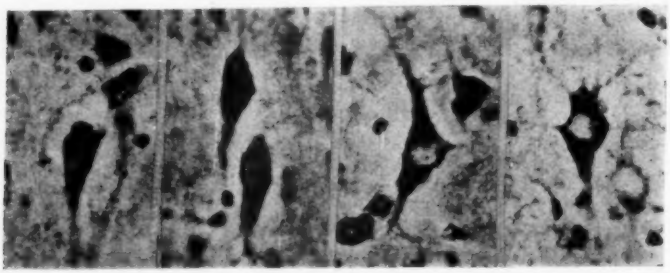


FIGURE V. Photomicrographs showing the effect of hypertonic (25%) glucose solution on the nerve cells. (Silver carbonate.)

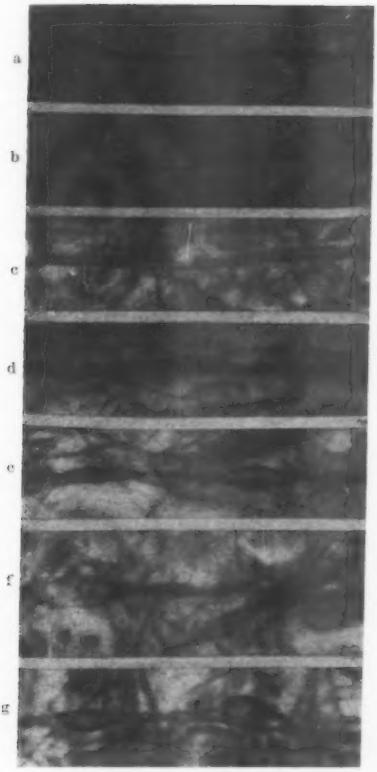


FIGURE VI. Photomicrographs showing swelling of the myelin sheaths. a, b, c: normal myelin sheaths; d, e: moderate swelling after the injection of distilled water with the skull intact; f, g: marked swelling when the bone and dura were removed prior to the injection of distilled water. (Weigert-Pal.)

between the wall and the cell contents. This continued until the cell wall ruptured, resulting in complete disintegration of the whole cell. The nuclei were swollen and they too were eventually destroyed. The Nissl granules showed little change, except that they became disarranged and compressed at one end of the cell. There was a distinct similarity between the process of swelling in the neurones and that found in the oligodendroglia, as will be shown when the latter cells are considered (Figure X). When glucose solution was injected, the nerve cells became greatly shrunken, leaving clear spaces around them (Figure V). Their nuclei appeared smaller than usual, but the Nissl granules showed little change other than being more closely packed together.

Myelin Sheaths.—There was moderate swelling of the myelin sheaths after the injection of distilled water with the skull intact (Figure VI, d, e). When the calvarium and dura were removed this swelling was intensified (Figure VI, f, g). In mild cases the swelling was patchy, with apparently normal areas in between the swollen parts. In more severe cases the swelling extended along the whole course of the sheath, with frequent bulbous enlargements. No evidence of swelling of the myelin sheaths was found in cases of cerebral œdema in man, and no change was noted after the instillation of 25% glucose solution.

Oligodendroglia.—These cells, of all the cerebral elements, proved to be the most susceptible to the presence of increased fluid within the tissues. This corresponds with the findings in cases in man in which the oligodendroglia were usually the first and frequently the only element to show evidence of swelling (Figure VIII). After the injection of distilled water with the skull intact, moderate swelling of the oligodendroglia was

FIGURE VII. Normal oligodendroglia. Note the relatively large nucleus and small amount of cytoplasm and the long delicate processes. (Silver carbonate.)

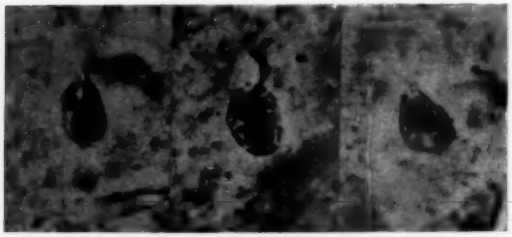


FIGURE VIII. Human oligodendroglia showing fairly marked swelling and rupture of the cell membrane. (Silver carbonate.)

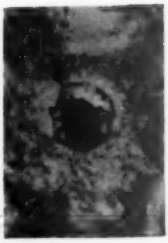


FIGURE IX. Swelling of the oligodendroglia of the cat after the injection of distilled water with the skull intact. (Silver carbonate.)

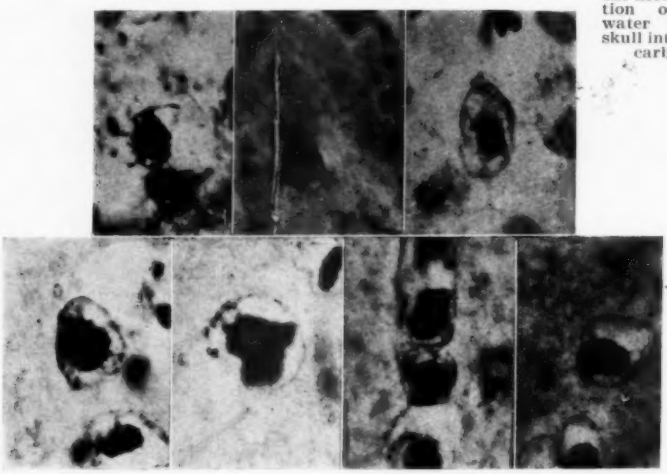
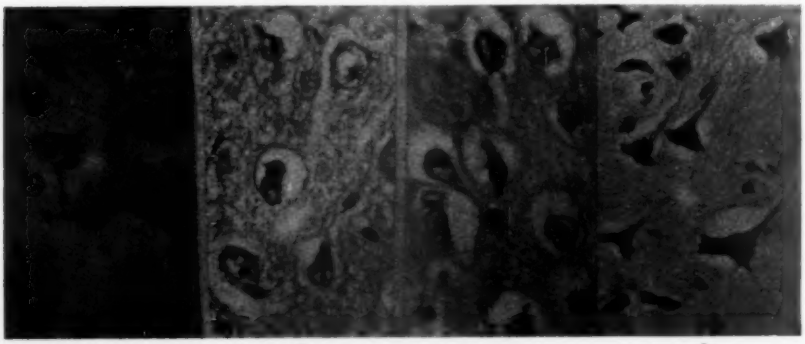


FIGURE X. Photomicrographic series demonstrating the progressive stages of swelling of the oligodendroglia when the calvarium and dura were removed prior to the injection of distilled water. Note the similarity to the changes in the nerve cells (Figure IV). (Silver carbonate.)



a b c d

FIGURE XI. Photomicrographs of the perineural spaces. a: normal; b: moderate dilatation after the injection of distilled water with the skull intact; c: marked enlargement after the injection of distilled water with the calvarium and dura removed; d: apparent dilatation caused by the shrinkage of the nerve cells after the injection of 25% glucose solution. (Hæmatoxylin and eosin.)

found throughout the sections (Figure IX). This was approximately of the same degree as has been found in cases of cerebral œdema in man, and may be compared with the normal cells shown in Figure VII.

When distilled water was injected, after removal of the calvarium and dura, the swelling of the oligodendroglia was more generalized and much more pronounced. These changes closely resembled those seen in the nerve cells under the same conditions, and all stages were seen from very early swelling to rupture and destruction of the cells (Figure X). In the early stages of œdema the delicate processes became fragmented, shortened and soon disappeared. As the intracellular fluid increased, the nuclei also

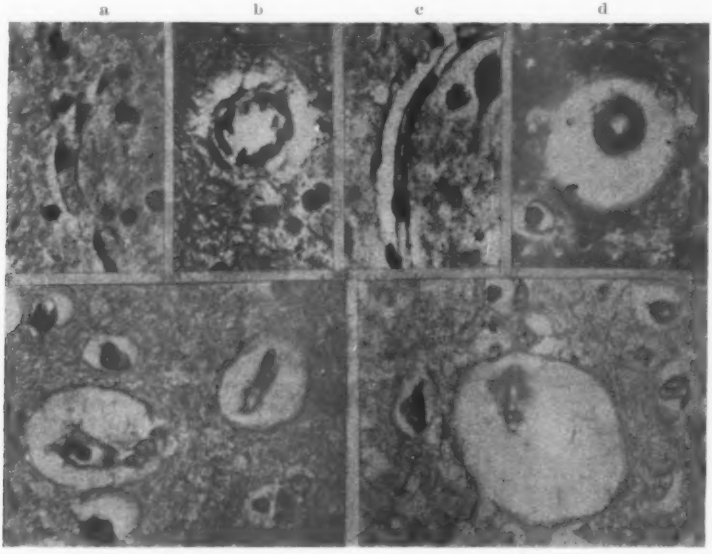


FIGURE XII. Photomicrographs of the perivascular spaces. a, b: normal; c, d: slight enlargement after distilled water with the skull intact; e, f: marked distension when the bone and dura were removed prior to the injection of distilled water. (Hæmatoxylin and eosin.)

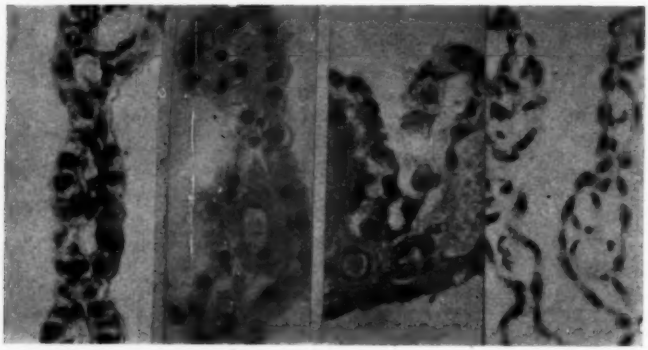
increased in size and were surrounded by clear areas which were bridged by fine fragments of cytoplasm. The cell became more swollen until eventually it ruptured and became completely disintegrated.

Perineural Spaces.—Slight increase in the perineural spaces was found when distilled water was injected with the skull intact (Figure XI, b). When the calvarium and dura were removed prior to the injection, the distension of the perineural space was much more marked (Figure XI, c). In the tissue that was exposed to the hypertonic effect of the 25% glucose, the apparent widening of the perineural spaces was really due to the shrinkage of the nerve cells (Figure XI, d). In human cases a mild degree of distension of the perineural spaces was found in the œdematous areas.

Blood Vessels.—No changes were found in the vessel walls. The Virchow-Robin spaces were distended after the injection of distilled water with the skull intact (Figure XII, c, d) and this was much more pronounced in those animals in which the calvarium and dura were removed prior to the injection (Figure XII, e, f,). No changes were found following the injection of 25%

glucose. In man there was usually some degree of widening of the perivascular spaces, more noticeable around the smaller vessels and capillaries.

Chorioid Plexus.—Moderate swelling of the lining cells of the chorioid plexus was found in those animals whose skulls were left intact during the injection of distilled water (Figure XIII, b). The nuclei were not enlarged, but were often eccentric in position. When the calvarium and dura were removed before the procedure, the swelling was intensified (Figure XIII, c). Following the injection of 25% glucose solution there was an extensive shrinkage of the chorioid plexus cells (Figure XIII, d). In most areas they were reduced to a thin, flat layer covering the chorioid vessels and connective tissue. In man these cells were frequently slightly larger than normal, and their nuclei tended to be situated somewhat nearer the periphery of the cell.



a b c d

FIGURE XIII. Photomicrographs of the lining cells of the chorioid plexus. a: normal; b: very slight swelling of the cells with a tendency towards an eccentric disposition of the nuclei, after distilled water with the skull intact; c: more evident swelling of the cells when the calvarium and dura were removed before the experiment; d: marked constriction of the cells after the injection of 25% glucose solution. (Hæmatoxylin and eosin.)

Ependyma.—In some areas there appeared to be slight swelling of the ependymal cells, but this was very inconstant even in the same section. For this reason no definite swelling of the ependyma can be reported from these experiments, and no changes in these cells were found in the human cases.

Astrocytes, Microglia, Axis Cylinders, Connective Tissue and Meninges.—No changes were observed in astrocytes, microglia, axis cylinders, connective tissue or meninges even in cases of severe œdema. The astrocytes and microglia showed no variation in size or shape, and their processes remained unaltered. The subarachnoid spaces contained less fluid than usual, but the actual meningeal tissue showed no microscopic change. These tissues showed no abnormal changes in human cases of cerebral œdema.

TISSUE REACTION AROUND EXPANDING LESIONS.

In this section the pathological changes found in the cells and supporting structures around neoplasms and inflammatory lesions are discussed. These changes were much more extensive and involved more elements than were involved in cases of pure cerebral œdema. Material for microscopic study was obtained from 28 of the human autopsy specimens, including 20 cases of neoplasm, five cases of meningitis, two cases of cerebral abscess, and one case of intracerebral hæmorrhage.

Evidence of œdema was invariably found; but, in addition to this, there was actual destruction of the tissues, and there were curious abnormal types of cells present which were not found in pure cerebral œdema. Whenever it was possible to take satisfactory photomicrographs reproductions have been made, and these are submitted to illustrate the various points under consideration.

Neurones.—The nerve cells in the immediate vicinity of an intracranial expanding lesion showed various stages of disintegration. In the early stages of destruction there was frequently an increase in the number of perineural satellites. In *astrocytoma diffusum* this was particularly noticeable, and many of the nerve cells showed no change, even when they were completely surrounded by tumour cells. The greatest destruction was found around acute inflammatory lesions and around the more rapidly growing neoplasms, such as *glioblastoma multiforme*. No changes were found in these cells resembling those seen in pure œdema, and there was no abnormal neurone formation.

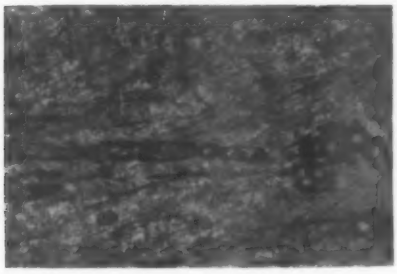


FIGURE XIV. Photomicrograph showing the changes found in the myelin sheaths in the neighbourhood of an intracerebral expanding lesion. (Weigert-Pal.)

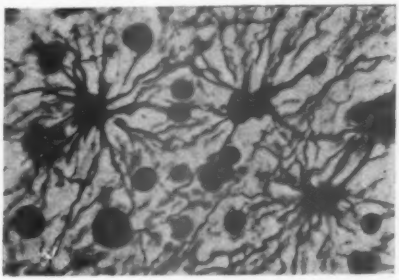


FIGURE XV. Photomicrograph of reactionary fibrous astrocytes in the tissue around an expanding lesion. Note the increased number of processes. (Silver carbonate.)

Myelin Sheaths.—Marked swelling of the myelin sheaths was found around neoplasms and abscess cavities, particularly in the case of the latter (Figure XIV). The swelling was frequently accompanied by the formation of small vacuoles within the sheaths. These changes became less pronounced the further the tissue was removed from the actual lesion. In the tissue closer to the lesion the myelin was more swollen and there was evidence of actual destruction and absorption by phagocytes.

Axis Cylinders.—The only change noted in the axis cylinders was one of destruction and absorption. They were usually completely destroyed for a varying distance around the lesion, but there was no evidence of actual swelling.

Astrocytes.—The most prominent cellular changes around intracerebral expanding lesions occurred in the astrocytes, and there was considerable variation in the type of cell seen. In some areas numerous fibrous astrocytes were found with a large number of fibrillary processes (Figure XV). These cells were probably not neoplastic in nature, but were produced as the result of tissue reaction in an attempt to form a barrier between the lesion and the surrounding brain. They were more commonly found around slow-growing neoplasms and walled-off abscess cavities. In the slow-growing lesions the cell body of the astrocytes eventually disappeared, leaving the fibres which became matted and compressed together to form a dense glial meshwork around the lesion.

In other situations the astrocytes showed considerable alteration in size and shape, a process which is degenerative in nature and is known as

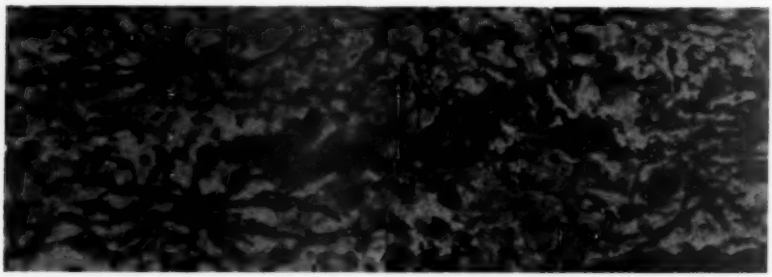


FIGURE XVI. Photomicrograph demonstrating the process of clasmatadendrosis. The processes become irregular, shortened and thickened, finally become fragmented and are removed as tissue débris along with the remains of the damaged cells. (Silver carbonate.)

"clasmatadendrosis" (Figure XVI). The cell processes became thickened, irregular in size and shape, twisted and eventually fragmented. The cell body also became irregular in shape, contracted and then broken up into fragments which were taken up and removed by phagocytes.

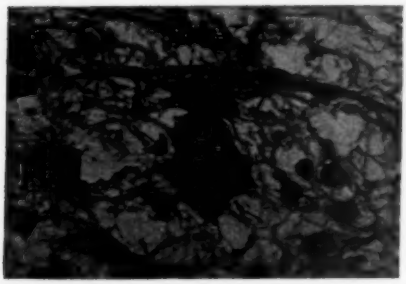


FIGURE XVII. Photomicrograph of Nissl's plump cells (*gemastëte Zellen*). (Hæmatoxylin and eosin.)

A third type of change noted in the astrocytes was not infrequently found around neoplastic and inflammatory lesions. These cells are referred to as Nissl's plump cells or *gemastëte Zellen* (Figure XVII). When they are found around these lesions they are usually reactionary in nature, but occasionally they are neoplastic in origin and form the type cell of one variety of astrocytoma. They are large, plump, thin-walled cells with short delicate irregular processes, and they do not contain fibres.

Oligodendroglia.—The type of change found in oligodendroglia was identical with that found in pure cerebral œdema, the only difference being one of degree (Figure XVIII). The progressive stages appeared to be the same, in that the delicate processes became fragmented and shortened, and the cells eventually ruptured and became absorbed by phagocytes. The pathological changes in the oligodendroglia were found over a much wider range than in the other tissues, and usually various stages of swelling were observed in those parts of the brain farthest removed from the primary lesion.

Microglia. — In contrast to pure cerebral œdema, marked changes in the microglia were observed around

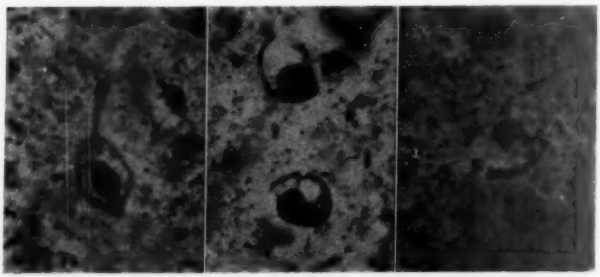


FIGURE XVIII. Photomicrographs showing marked swelling of the oligodendroglia around intracerebral expanding lesions. (Silver carbonate.)

certain intracerebral expanding lesions. This was particularly noticeable around inflammatory lesions, areas of necrosis and cerebral softening, and around areas of degeneration in neoplasms. The microglia cells (Figure XIX) are homologous to the cells of the reticulo-endothelial system. They are phagocytic in nature and, in inflammatory and necrotic processes, they proliferate and change their form to become what are known as compound granular corpuscles (Figure XX). These cells are round or ovoid with small eccentric nuclei, and are filled with granules and tissue débris which they remove from the site of disease to the neighbouring blood vessels. When all the diseased tissue has been removed and replaced by glial tissue these cells disappear.

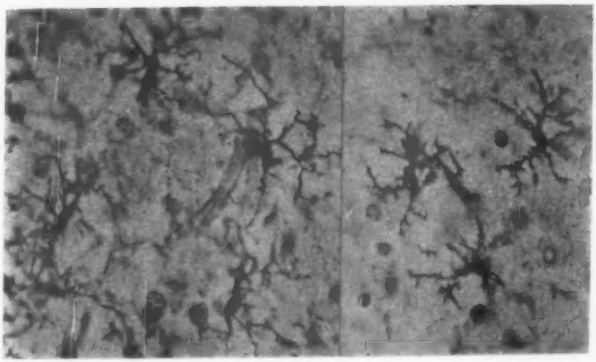
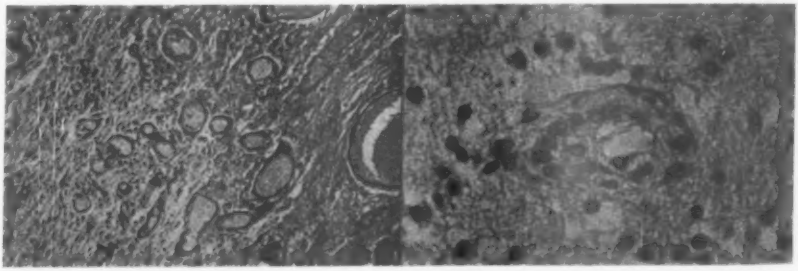


FIGURE XIX. Photomicrographs of normal microglia. (Silver carbonate.)

Ependyma.—The ependymal cells showed no change, except in those cases in which the lesions were closely approximated. When this occurred, the cells became slightly swollen. When they were actually involved in the pathological processes, they were completely destroyed.

Chorioid Plexus.—Little change was observed in cells of the chorioid plexus unless they were actually involved in an inflammatory or neoplastic process. When this occurred the cells became swollen, and were eventually destroyed and removed by phagocytes.

FIGURE XX. Photomicrograph of a group of compound granular corpuscles. (Hæmatoxylin and eosin.)



a b

FIGURE XXI. Photomicrographs showing the changes found in the blood vessels around expanding lesions. a: increased formation of fine capillaries; b: proliferation of the endothelial cells in the lumen of a vessel in the vicinity of a *glioblastoma multiforme*. (Hæmatoxylin and eosin.)

Blood Vessels.—The blood vessel reaction varied considerably in different lesions. In the vicinity of acute inflammatory lesions there was little change except that the lumen and perivascular spaces were often filled with neutrophile cells. In the neighbourhood of chronic inflammatory lesions and certain neoplasms there was a definite increase in the number of tiny blood vessels (Figure XXI, a). In the case of neoplasm these newly formed blood vessels eventually became surrounded by tumour cells. Frequently the tiny vessels ruptured and gave rise to varying sized hæmorrhages. One point is worthy of note regarding the appearance of newly formed blood vessels in the advancing borders of a *glioblastoma multiforme*: the lining endothelial cells showed marked proliferation, frequently to the extent of completely filling the lumen of the vessel (Figure XXI, b). This phenomenon is almost pathognomonic of this type of glioma.

Connective Tissue.—There was usually some degree of mesodermal reaction around chronic inflammatory lesions and certain of the more slowly growing neoplasms (Figure XXII). The connective tissue is derived from the blood vessels, and a dense barrier is eventually formed around the lesion. The fibrous tissue seen around neoplasms usually consists of a few irregular scattered strands, although frequently it forms a fairly complete capsule around such lesions as metastatic carcinoma. The perineoplasmic connective tissue can frequently be seen extending inwards between the tumour cells, where the process of connective tissue formation continues. Both collagenous and fibrous connective tissue may be seen, but in the older lesions little or no collagen fibres can be found.

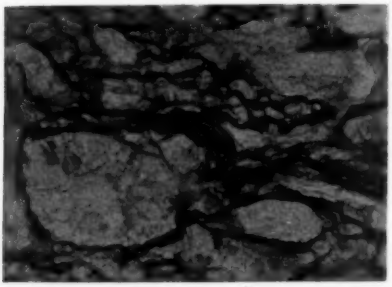


FIGURE XXII. Photomicrograph showing the overgrowth of connective tissue seen around certain types of intracerebral expanding lesion. (Laidlaw's connective tissue stain.)

Meninges.—No changes were found in the meninges, unless they were actually involved in an inflammatory or neoplastic process. In such cases they became thickened and the normal relationship between dura, arachnoid and pia was lost. Additional connective tissue was laid down, and inflammatory or neoplastic cells could be found lying between the strands.

TREATMENT.

This survey would not be complete without a word in regard to the treatment of cerebral œdema. The object of the treatment is threefold, and should be directed toward: firstly, the eradication of the primary lesion, whether it be intracranial or extracranial; secondly, the relief of the increased intracranial pressure; and, thirdly, the removal of the excess intracerebral fluid.

In the majority of cases it is sufficient to remove the primary lesion without directing any special attention towards the accompanying œdema. In pronounced œdema following head injury or intracranial surgical procedures, and in severe cases of toxic œdema, it is necessary to attempt to withdraw some of the excess intracerebral fluid. In a hæmorrhage caused by head injury the blood and clot should be evacuated and any septic focus, in the case of toxic œdema, should be treated. The intracranial pressure is relieved by lumbar drainage of the cerebro-spinal fluid. Care must be taken not to reduce the pressure by lumbar puncture unless the surgeon is absolutely

certain that there is no space-occupying lesion within the cranial cavity. If an expanding lesion is present and spinal fluid is removed carelessly by the lumbar route, there is grave danger of the brain herniating through the *incisura tentorii* or through the *foramen magnum*, as has been shown by Reid and Cone.[16][17]

In certain cases lumbar drainage may be the only method possible, and in such instances the risk of herniation is lessened by attaching a rubber manometer to the needle and the outlet to a stand. The outlet is placed at the position of the initial pressure and then lowered an inch about every ten minutes. In this manner the pressure is gradually reduced to normal over a period of one to one and a half hours. Even with this method, however, there is some risk of herniation. If there is no intracranial focal expanding lesion, there is less risk in lumbar drainage, even in cases of very high pressure, but it is wise to reduce the pressure slowly. Lumbar drainage should be repeated as often as is necessary to maintain the pressure at a normal level and until it remains within normal limits.

If the preceding two methods are not sufficient to relieve the patient's condition, it is necessary to attack the œdema directly. This may be done by giving the patient repeated intravenous injections of hypertonic glucose or sucrose solutions and by the administration of diuretics. Sucrose in a 25% or 50% solution is the most suitable substance, as it has certain advantages over glucose.

Milles and Hurwitz[18] found that glucose was lost from the blood stream by excretion through the kidneys and by removal into the tissue spaces and cerebro-spinal fluid. The osmotic pressure of the cerebro-spinal fluid soon became higher than that of the blood, and the process was reversed, giving a secondary rise in the cerebro-spinal fluid pressure, which reached a point much higher than the original level. Gregersen and Wright[19] reported that the intravenous injection of three to six grammes of sucrose per kilogram of body weight did not produce any measurable increase in the sugar content of the cerebro-spinal fluid. When they injected 3·0 to 3·5 grammes of glucose per kilogram of body weight, they found a marked hyperglycorrhachia up to 187 milligrammes per 100 cubic centimetres (normal = 50 to 75 milligrammes *per centum*).

The use of diuretics is frequently of value in eliminating cerebral œdema, the most useful of these being "Salgyran". This drug is given intramuscularly or intravenously in a dosage of 0·5 to 2·0 cubic centimetres every two or three days. It should not be given in cases of renal disease or cachexia. I have found this drug very effective when combined with intravenous injections of hypertonic sucrose solution. A test dose of 0·5 cubic centimetre of "Salgyran" is given, and then one cubic centimetre every two to three days for one to two weeks. The urine is examined daily during this period for the presence of albumin and blood, and if either of these is found the drug should be discontinued. At the same time 200 cubic centimetres of 25% solution of sucrose are given intravenously over a period of thirty to forty minutes once or twice a day. It is advisable to avoid too rapid or too violent dehydration, as occasional reports have been made of nephritis following sucrose therapy. I have found this method of treatment especially effective in post-traumatic œdema. I have not used "Salgyran" in œdema of toxic origin because of the possible presence of renal damage; but I have no evidence that it is detrimental in such cases. In very severe cases, especially those of toxic origin, a large bilateral subtemporal decompression is of great value in alleviating the patient's symptoms.

The routine adopted in the treatment of cerebral œdema varies in each case and depends to a large extent on the cause and severity of the œdema. In mild cases the treatment of the cause is usually sufficient to induce the œdema to abate of its own accord. In more severe cases active treatment should be adopted in the form of cerebro-spinal fluid drainage, intravenous injections of hypertonic glucose or sucrose solutions, diuretics and subtemporal decompression.

DISCUSSION.

The object of this study has been primarily to present a clear picture of the pathological changes found in cerebral œdema resulting from an excess of fluid within the cerebral tissues. In addition to this, an attempt has been made to show that excess tissue fluid alone cannot produce the histological changes that are found in the tissues around intracranial expanding lesions.

A series of experiments has been carried out in seventeen cats, of which four were used as control animals. Œdema of the cerebral tissues was produced by instilling distilled water into the cerebral circulation. In some of the animals the skull was left intact during the procedure so that the maximum degree of œdema obtainable in these circumstances could be produced. In others the calvarium and dura were removed prior to the commencement of the experiment. This was done to permit the brain to absorb as much fluid as possible, unrestricted by the overlying bone and dura. It was assumed that any cellular or supporting structure that could be influenced by excess fluid would exhibit histological changes under these conditions. Conversely any tissue that did not reveal some histological change under these conditions would not show any reaction in ordinary human œdema.

To obtain a clear picture of the pathological changes that occurred in human cerebral œdema, 47 cases were studied at autopsy. These comprised 16 cases of trauma, in which death occurred several hours after the head injury; 20 cases of neoplasm; five cases of meningitis; three cases of extradural hæmorrhage; two cases of cerebral abscess and one case of intracerebral hæmorrhage. Sections were taken from parts of the brain furthest removed from the site of the lesion. The pathological findings were similar in each case, the only difference being one of degree.

The most marked changes were found in acute inflammatory lesions and in large rapidly expanding neoplasms. The macroscopic appearance of cerebral œdema was similar in human cases and in the laboratory animals, again the only difference being one of degree. In mild cases the gyri were slightly flattened and the sulci narrowed. This was probably due mainly to compression of the brain surface against the inner table of the skull by the increased intraventricular pressure, as it was much less evident when the skull and dura were removed prior to the experimental procedures. The subarachnoid spaces contained less fluid than normal—a feature which was probably also the result of compression. The cut surface was moister than usual and had a rather glazed appearance.

In cases of cerebral œdema in man the oligodendroglia, perineural and perivascular spaces and the lining cells of the chorioid plexus showed histological changes. No alterations were found in any of the cases in the myelin sheaths, nerve fibres, astrocytes, microglia, ependyma, connective tissue or meninges. The nerve cells only very occasionally showed any evidence of swelling in the more severe cases of œdema. The nuclei of these cells were slightly larger than normal and had a somewhat glazed appearance. These changes were so inconstant that it is doubtful whether they should be regarded as a definite part of the picture of cerebral œdema.

The oligodendroglia showed the most definite and constant histological changes. In every case in which the examination was made these cells showed evidence of swelling. In small slowly growing lesions the swelling was mild and did not involve all the cells. In large rapidly expanding lesions and in acute inflammatory lesions the swelling was marked, more uniform and frequently resulted in the rupture and complete disintegration of the cell.

There was usually some distension of the perineural and perivascular spaces. These features were not constant in small expanding lesions, but were quite definite in acute inflammatory lesions and in large rapidly growing neoplasms. In the more severe cases the lining cells of the chorioid plexus were usually larger than normal and their nuclei tended to be eccentric in position.

In those laboratory animals in which the skull was left intact, the same cellular and supporting structures were affected as in human œdema, with the addition of the nerve cells and myelin sheaths. The only difference was one of degree, in that the swelling was more pronounced in the animals. Occasional nerve cells were a little larger than normal, and their nuclei were slightly distended and had a glazed appearance. The changes in the myelin sheaths were more definite, the swelling occurring at intervals along the course of the nerve fibres.

When the calvarium and dura were removed prior to the injection of the distilled water, the same structures were affected, but the degree of œdema was much more intense. The nerve cells and oligodendroglia showed all stages of swelling, resulting in rupture and complete disintegration. The myelin sheaths were distended all along their course. The perineural and perivascular spaces were greatly enlarged, and in addition to these the lining cells of the chorioid plexus were swollen.

It is very significant that in no instance did the astrocytes, microglia, nerve fibres, connective tissue or meninges show any evidence of swelling, even in the most severe experimental œdema. Some of the ependymal cells appeared to be swollen in a few of the sections, but this was not sufficiently constant to be regarded as a definite feature. From the data obtainable in this study there does not seem to be any indication to subdivide this condition into "cerebral œdema" and "brain swelling". The changes were the same in type in every case, the only variation being one of degree. The picture was that of an excess of fluid within certain cells and intracellular tissues. The resulting œdema was slight in cases of small slow-growing lesions and most marked in acute inflammatory lesions and in large rapidly growing neoplasms. The type of tissue involved in the œdema process was the same irrespective of the nature of the causative lesion.

The changes found in the cellular and supporting structures around intracerebral expanding lesions were very different to those found in pure cerebral œdema and practically all the tissue elements were affected. There was evidence of œdema, but besides this there was considerable destruction of the tissues and certain abnormal cell forms made their appearance; but these were not seen in pure œdema.

The nerve cells and the axis cylinders showed little change, except that of destruction by the pathological process. There was no actual swelling in these two tissues. The oligodendroglia and myelin sheaths were greatly swollen, more so than in the case of pure œdema. The oligodendroglia showed all stages of swelling to rupture and complete disintegration of the cells. In the myelin the swelling was pronounced and was accompanied by the formation of vacuoles within the sheaths, and the myelin was eventually broken down and removed by phagocytes.

The most prominent changes occurred in the astrocytes and microglia, which cells remained unaffected in pure cerebral œdema. Large numbers of reactionary fibrous astrocytes were found with an increased number of fibrillary processes. In very slow-growing lesions these formed a barrier between the lesion and the brain tissue, and eventually the cell bodies disappeared, leaving a dense meshwork of intertwining glial fibres. In other areas one found the process of clasmatadendrosis in which the astrocyte fibres became thickened, irregular and fragmented, and eventually, along with the cell body, they were removed by phagocytes. The third type of astrocyte change was seen in the formation of Nissl's plump cells or *gemastëte Zellen*. These were large, fat cells, with short, delicate processes and no fibres.

The microglial change was even more remarkable. These cells lost their processes, became shorter and rounder, and eventually assumed a round or ovoid shape with granular cytoplasm. They became the active phagocytes of the brain to remove much of the tissue débris to the neighbouring blood vessels, and in this form are known as compound granular corpuscles. These cells were never found in pure cerebral œdema.

The blood vessels themselves also showed definite changes. In some areas there was a marked increase in the number of small blood vessels. In certain lesions, particularly around the margins of *glioblastoma multiforme*, there was a curious phenomenon, the exact cause of which is not fully understood. This consisted of a marked proliferation of the endothelial lining cells, sometimes to the extent of completely obliterating the lumen of the vessels. The connective tissue was usually increased around the margins of intracerebral expanding lesions. This connective tissue was derived from the blood vessels, and varied from a few scattered strands to a complete well-formed fibrous capsule, as is found around some chronic abscess cavities.

No definite changes were found in the cells of the ependyma or chorioid plexus, or in the meninges, unless these structures were actually involved in the primary pathological process. In such cases the cells usually showed slight swelling, and were eventually completely destroyed. The meninges became thickened and irregular, and there were frequently large numbers of compound granular corpuscles in the subarachnoid spaces.

With reference to the various methods of treatment that may be adopted, the main objects are to relieve the intracranial pressure, which may become very high, and to remove the excess intracerebral fluid in order to prevent cerebral anoxæmia of the parts affected. The former is done by the eradication of the causative lesion, by lumbar drainage of the cerebro-spinal fluid and by unilateral or bilateral subtemporal decompression. The latter is effected by intravenous injection of hypertonic glucose or sucrose solutions and by the use of diuretics. The fluid intake should not be restricted too much, as this is of little value unless the restriction is considerable and such a course may produce extreme dehydration, which would do more harm than good.

SUMMARY AND CONCLUSIONS.

The conclusions that have been drawn from the data obtained from the experimental procedures and from the human cases are as follows:

1. Cerebral œdema can be produced by brain trauma due to head injuries, operations or expanding lesions, by intracranial inflammatory lesions and by toxins produced from extracranial septic foci and certain poisons.

2. In human cerebral œdema the gyri are flattened and the sulci narrowed. There is less fluid in the subarachnoid spaces than usual, and the cut surface of the brain is moist and has a rather glazed appearance. The

oligodendroglia always show various degrees of swelling. The perineural and perivascular spaces are widened and in severe cases the lining cells of the chorioid plexus are slightly swollen with somewhat eccentric nuclei. No changes occur in the axis cylinders, myelin sheaths, astrocytes, microglia, ependymal cells, connective tissue or meninges. The nerve cells occasionally show signs of mild swelling, but this is inconstant.

3. In the vicinity of intracerebral expanding lesions almost all the cerebral elements are affected. The nerve cells and the axis cylinders show only slight evidence of destruction. The oligodendroglia and myelin sheaths become greatly swollen, eventually rupture and are removed by phagocytes. Astrocyte changes occur: (*a*) in the formation of fibrous astrocytes with numerous fibres forming a barrier between the lesion and the brain, the cell bodies disappearing, leaving a dense glial meshwork; (*b*) in the production of *gemastëte Zellen*; and (*c*) in a curious method of destruction known as clasmatadendrosis. The microglia are converted into compound granular corpuscles which become phagocytes to remove the damaged tissue. The blood vessels may be increased in number, and in certain cases there is a proliferation of their endothelial cells. The perineural and perivascular spaces are usually distended, and the latter are often filled with compound granular corpuscles. Connective tissue strands extend outwards from the blood vessels, and frequently this is quite dense. The chorioid plexus cells, ependyma and meninges usually show no change unless they are directly involved in the pathological process.

4. The changes observed around intracerebral expanding lesions are obviously due to something more than a simple excess of intracerebral fluid.

REFERENCES.

(1) Paulo Pupo: "Role of Cerebral Edema in the Pathogenesis of Convulsions", *Arq. Serv. assist. psicopat. estad. Sao Paulo*, Volume v, 1940, page 16.

(2) H. Spatz: "*Die Bedeutung der symptomatischen Hirnschwellung für die Hirntumoren und für andere raumbeengende Prozesse in der Schädelgrübe*", *Archiv für Psychiatrie und Nervenkrankheiten*, Volume lxxxviii, 1929, page 790.

(3) I. Schienker: "Cerebral Swelling and Edema Associated with Cerebral Tumor: A Histogenic and Histopathologic Study", *Archives of Neurology and Psychiatry*, Volume xlv, 1941, page 117.

(4) M. de Crinis: "*Ueber die Diagnose und Behandlung de Hirnschwellung*", *Weiner klinische Wochenschrift*, Volume liii, 1940, page 637.

(5) L. B. Rawling: "Cerebral Œdema (Excess Cerebro-Spinal Fluid): Its Causation and Surgical Treatment", *British Medical Journal*, Volume i, 1918, page 499.

(6) M. Reichardt: "*Über die Bestimmung der Schädelkapazität an der Leiche*", *Allgemeine Zeitschrift für Psychiatrie*, Volume lxii, 1905, page 787.

(7) L. Jaburek: "*Hirnödem und Hirnschwellung bei Hirngeschwülsten*", *Archiv für Psychiatrie und Nervenkrankheiten*, Volume civ, 1935-1936, page 518.

(8) A. Cantarrow: "Water Balance: Edema and Dehydration: A Review of Recent Progress", *The New International Clinic*, Volume i, 1939, page 266.

(9) W. Penfield and W. V. Cone: "Acute Swelling of Oligodendroglia: A Specific Type of Neuroglia Change", *Archives of Neurology and Psychiatry*, Volume xvi, 1926, page 131.

(10) W. Penfield and W. V. Cone: "The Acute Regressive Changes of Neuroglia (Amœboid Glia and Acute Swelling of Oligodendroglia)", *Journal für Psychologie und Neurologie*, Volume xxxiv, 1926, page 204.

(11) C. W. Apfelbach: "Studies in Traumatic Fractures of the Cranial Bones. 1. Edema of the Brain. 2. Bruises of the Brain", *Archives of Surgery*, Volume iv, 1922, page 434.

(12) P. Shapiro and H. Jackson: "Swelling of the Brain in Cases of Injury to the Head", *Archives of Surgery*, Volume xxxviii, 1939, page 443.

(13) C. Pilcher: "Experimental Cerebral Œdema: The Fluid Content of the Brain after Trauma to the Head", *Archives of Surgery*, Volume xxxv, 1937, page 512.

[14] L. H. Weed and P. S. McKibben: "Experimental Alteration of Brain Bulk", *American Journal of Physiology*, Volume xlviii, 1919, page 531.

[15] J. G. Greenfield: "The Histology of Cerebral Œdema Associated with Intracranial Tumours (with Special Reference to Changes in the Nerve Fibres of the Centrum Ovale)", *Brain*, Volume lxii, 1939, page 129.

[16] W. Lister Reid and W. V. Cone: "The Mechanism of Fixed Dilatation of the Pupil Resulting from Ipsilateral Cerebral Compression", *The Journal of the American Medical Association*, Volume cxii, 1939, page 2030.

[17] W. Lister Reid: "Cerebral Herniation Through the Incisura Tentorii", *Surgery*, Volume viii, 1940, page 756.

[18] G. Milles and P. Hurwitz: "The Effect of Hypertonic Solutions on Cerebro-Spinal Fluid Pressure, with Special Reference to Secondary Rise and Toxicity", *Archives of Surgery*, Volume xxiv, 1932, page 591.

[19] M. I. Gregersen and L. Wright: "The Effect of Intravenous Injection of Sucrose and Glucose upon the Reducing Power of the Cerebro-Spinal Fluid, Before and After Hydrolysis", *American Journal of Physiology*, Volume cxii, 1935, page 97.

ABDOMINAL SURGERY IN THE ALAMEIN CAMPAIGN.[1]

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THIS paper is based on the experience gained from operating on 90 patients with abdominal wounds in forward areas at Alamein during the period July to November, 1942. The work was done at the main dressing stations of the field ambulances of the Ninth Division, by an Australian Imperial Force team sent up by an Australian general hospital. Personnel of the team was one surgeon, one assistant surgeon (used as an anæsthetist if necessary), three operating-room assistants and two drivers. Two three-ton trucks carried adequate equipment. An electric generator and shadowless light, a rubber floor covering, especially designed theatre furniture[2] and a foot suction pump facilitated work and permitted planned rather than makeshift operation, even when working in an "E.P.I.P." pattern tent some twelve by fifteen feet.

The high proportion of abdominal cases—90 in a total of 220—was to a great extent the result of the static warfare during this period and to the presence of casualty clearing station facilities some four hours further back. In these circumstances it was justifiable to give abdominal wounds, even the very severe ones and the doubtful risks, high priority. With the exception of those patients with severe hæmorrhage and those with tourniquets, few of the other wounded deteriorated from delay in surgical treatment.

The surgery of abdominal cases naturally fell into two phases: the first —Phase 1—during the earlier half of the tour of duty when orthodox methods were followed; and the second—Phase 2—when, with experience gained in the early surgery of Phase 1, new standards of resuscitation, pre-operative and post-operative treatment were set and new methods of operation were adopted.

The results of Phase 2 give an indication of the minimum recovery rate that should be expected in the future from a surgical unit adequately equipped for abdominal surgery, receiving patients within twelve hours, and following the principles advocated for pre-operative and post-operative care and the procedures outlined for safe surgery in wounds of the colon.

At the end of the paper is an appendix which contains a brief summary of the histories of those cases to which reference is made in the text.

GENERAL MANAGEMENT.

Pre-Operative Management.—All patients were admitted to the resuscitation ward of the main dressing station, which was administered by the ambulance resuscitation officer, and to which the theatre of the surgical team was brigaded as an annexe. The patients were seen by the surgeon as soon as possible, and a complete examination of all wounds and a diagnosis as to the probability of peritoneal perforation were made. Any sign of peritoneal perforation was considered a definite indication to explore the abdomen as soon as the patient attained a fit condition. During periods in which odd casualties were coming in, the response of the patient to resuscitation could be watched by the surgeon himself, who, in consultation with the resuscitation

[1] Accepted for publication on May 25, 1943.

[2] Designed by Lieutenant-Colonel F. J. Clark, made of stainless metal, collapsible and taking little space in the trucks, and presented by the Australian Red Cross Society.

officer, could choose the optimum time for operation—that is, the time when the patient had reached his best general condition. But during battle, with many casualties coming into the resuscitation ward and with continuous operating, after patients had been seen at least once by the surgeon the "operation list" had to be left to the resuscitation officer.

RESUSCITATION.

Resuscitation in this article means the intravenous administration of fluids, such as blood, serum, plasma, and in rare cases saline solution. All but very few of our patients were in need of this intravenous therapy before operation. Although resuscitation was essentially under the control of a resuscitation officer of the field ambulance, to which the surgical team was attached, consultations were held on patients who were not responding to treatment in a reasonable time.

In our earlier cases, comprising patients received at a later period after being wounded, on account of the difficulties of evacuation by routes under gun-fire to a main dressing station situated some fifteen miles behind the fighting, several hours were sometimes spent in resuscitation. To these patients blood or serum was given at what was then thought to be a reasonable rate—that is, taking perhaps thirty to forty minutes to give over a pint. 'Soluvac" apparatus was used. Serum or banked blood, sent up by Number 1 Base Transfusion Unit in "English" bottles, was emptied into Australian "Soluvac" "giving sets". It was found that patients suffering a mild degree of shock requiring from 1,000 to 1,500 cubic centimetres of fluid, in general responded quickly and did not suffer from this "slow resuscitation" treatment.

This was far from being the case in two groups of patients—namely, those in whom large vessels were severed, and those suffering from severe shock, the result of a ruptured bowel. The first group showed little response to transfusion at this slow rate and were finally operated on as bad risks in order to control hæmorrhage. Two patients (Cases VII and XXXI) are typical examples of this. In the second group valuable hours were sometimes wasted in attempts to make the patient a good risk. In some cases the patient's condition improved and this end was attained, but in others his condition would improve to a point and then "hang fire". When these patients were operated on, a commencing or well established peritoneal reaction was found.

After a month the situation was reviewed, and it was decided that one and a half hours was the longest time that should be allowed for resuscitation, and that if there was little improvement in this time, operation should be performed. It was found that patients who did not respond in this period nearly always had either a fairly severe internal bleeding or multiple perforations, and in these cases there was little likelihood of any marked response with continued resuscitation.

"Rapid Resuscitation."—Captain R. Formby, resuscitation officer of a field ambulance to which the team became attached from early in August until November, determined to try more rapid methods of resuscitation in an endeavour to produce good risks for operation in this specified time. He introduced into the circulation large volumes of serum and blood in a very short time, but never saline or glucose saline solution. His standard procedure in the treatment of all moderately or severely shocked patients was the administration of 500 cubic centimetres of serum during the first five to ten minutes. If the patient was not very exsanguinated, he gave another 500 cubic centimetres in the next ten minutes, and then started the transfusion of blood. This was frequently given for the first two pints at the rate of one pint in ten minutes, and in a few cases at double this speed. It was

considered that any "giving set" that could not give a pint of blood in this time was inefficient for resuscitation in forward areas. The English "giving sets" were used as a routine and proved excellent. With them positive pressure could be applied to the bottle and a pint of blood could be given in five minutes. But when, on a few occasions, "Soluvac" bottles, filled with fresh blood taken from men in the division, were used, the rate of flow was disappointing when rapid transfusion was required and their use had to be given up. The filter in the Australian "giving set" is superior to that of the English, but in most other features, the size of the bottle and bulky pack, the unnecessarily complicated screw-top, the fixed glass tube and the impossibility of producing positive pressure in the bottle (leakage occurring round the top), it was inferior. And also, through the needles supplied, it is not possible to give blood at a sufficiently rapid rate.

The results of this "rapid resuscitation" surpassed all expectations. There were few patients, the moribund excepted, who had not a blood pressure stabilized at over 100 millimetres in under one and a half hours. This was the blood pressure set as the standard of a fair operative risk.

Similar volumes of blood or serum produced very different effects according to whether they were introduced slowly or rapidly. Not only a much more rapid, but also a greater, rise in blood pressure followed rapid introduction. This result was also observed in cases in which no further hæmorrhage was occurring, as, for example, limb wounds with tourniquets on. Once the blood pressure was raised to normal or low normal, it was found that it could be maintained at this level by a slow drip. It would appear that in severely shocked patients not only has the mechanical factor of replacement of fluid to be dealt with, but also the vasomotor mechanism, which acts on a "ratchet" principle; moreover, this can be most effectively stimulated into action by rapid introduction of fluids. The type of case that in our early experiences was found to give no response to "slow resuscitation", either because of severe continued bleeding or because of spreading infection or irreparable tissue damage from anoxæmia, with "rapid resuscitation" nearly always gave a response, although this was generally somewhat delayed.

Case XCIX illustrates the efficiency of this method of rapid transfusion.

A pulseless patient with nearly half the small intestine prolapsed and in tatters and with multiple shell wounds of both thighs, was admitted at 1235 hours. Transfusion was started and after about one hour the pulse was quite detectable at the wrist and bleeding from the collapsed gut became free. Transfusion was still further speeded and notwithstanding a blood loss estimated at one pint in twenty minutes, he gained in his general condition. This gain continued, and by 1545 hours, after having received 2,500 cubic centimetres of serum and 4,500 cubic centimetres of blood, he had a systolic blood pressure of 100 millimetres of mercury and a diastolic pressure of 50 millimetres. The operation included extensive resection of small intestine, ligation of the left deep femoral vessels, repairs to the abdominal wall, excision of extensive wounds in the right thigh. During operation a further 1,500 cubic centimetres of serum and 1,500 cubic centimetres of blood were administered. At the end of operation his systolic blood pressure was 90 and his diastolic pressure 50 millimetres of mercury.

In this rapid method of transfusion the rate was not slowed even if reaction to serum or blood occurred; but the whole "giving set" and its contained fluid were replaced and the speed of the transfusion was maintained. Reactions did not occur in any higher percentage of cases than when a slow drip was used. In no case was there any evidence of cardiac embarrassment because of this rapid introduction of fluid.

OPERATIVE TREATMENT.

Preparation for Operation.—In all cases cleansing was carried out with ether soap, the area was shaved and then the skin was prepared with

methylated spirit and iodine. If possible this was done in the resuscitation ward, but in many cases, particularly those with multiple wounds and fractures, it was necessary to do this in the theatre under anæsthesia.

General Management in the Theatre.—All patients had intravenous drips adjusted and running before coming to the theatre. At first, if their condition was satisfactory, this was not done. Experience showed, however, that most patients had far more intraperitoneal hæmorrhage than was anticipated, particularly in the case of lesions of the small bowel, and for this reason the patient's condition tended to deteriorate when the peritoneum was opened.

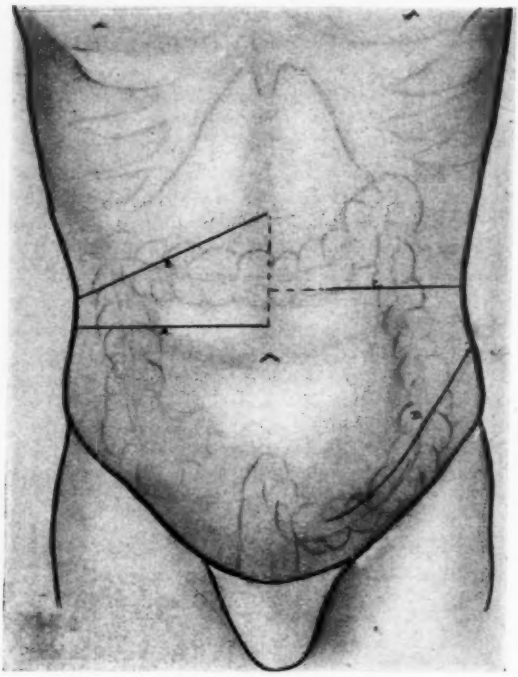

FIGURE I. Showing the various incisions used for the different types of abdominal injuries. A = transverse incision commonly made for all wounds in right renal flank or lateral areas. Dotted line shows extension upward (in mid-line) if foreign body had ascended intraperitoneally. Right colectomy *plus* nephrectomy easily performed through this without extension. B = modification (half-Kocher–half-transverse) found most useful in all wounds through lower part of the chest. C = a similar transverse incision to A on left side. Commonly used for suspected colon, kidney or retroperitoneal hæmatomata for left plane wounds. This has been left short of mid-line, as my records show that in this one, as opposed to A, it was often unnecessary. Any splenic or gastric lesion was adequately exposed by continuing to mid-line and then up on the dotted line. D = the long-low oblique incision used for wounds in the left iliac fossa or round the left hip joint or buttock. It has been made too near the mid-line.

The introduction of routine post-operative gastric suction and the intravenous administration of fluids in all cases of bowel damage was another reason for having a drip going even if there was no gross bleeding. Practically all patients left the table with no reduction in blood pressure because blood, serum or, at times, saline solution was used during operation.

Anæsthesia. — "Open" ether anæsthesia was used in practically all cases. No gas anæsthesia was carried by the team, nor was any need felt for it in these cases. Oxygen or "Carbogen" was frequently used by anæsthetists. Most of the patients had received large morphine injections, and in some cases respiration was fairly depressed. "Pentothal" was available, but was not used because anæsthesia in most abdominal operations was required for one and a half hours, and it was felt that in the presence of shock depression of respiration for this time would be bad. The anæsthetic was administered by a medical officer, for many of the patients were not good anæsthetic risks and the use of an orderly as anæsthetist in these circumstances would have raised the mortality rate.

Abdominal Incision.—The majority of wounds were from shell, shrapnel or bomb. In general, these were penetrating, with little indication of their

abdominal course or of the final site of the foreign body. Machine-gun and gunshot wounds were often perforating in type. X-ray equipment was not carried by the team, nor was it felt that its use would have been of help. If there were signs of intraabdominal injury, or even if there were doubtful signs, operation was considered necessary. Operation was to deal with intra-abdominal lesions, to drain extraperitoneal hæmatomata, and to excise grossly damaged or infected tissues. A search for and removal of foreign bodies, if not in the abdominal cavity or in association with bowel, was considered an unnecessary operation and was not done.

Flank or lateral abdominal wounds, of which there was a fairly high proportion, were generally dealt with by means of a transverse or slightly oblique incision (see Figure I). This gave direct access to the area of known damage and particularly to the fixed portions of the colon. The incision was made through the lateral half of the rectus muscle and the peritoneum was opened. If further access was required, the incision was extended to the mid-line and then upward or downward prolongations were made as required. The external oblique muscle was cut in the line of the incision; the internal oblique and transversalis muscles were split. In wounds through the lower ribs a similar transverse incision with an upward mid-line extension to the xiphisternum was used. Wounds in the iliac fossa or through the ilium were explored through a low oblique incision lateral to the rectus sheath. If it was necessary to inspect or mobilize the colon or caecum, this incision was extended laterally just above the ilium. For all wounds near the mid-line a vertical incision, either median or paramedian, was used. Tangential wounds of the anterior abdominal wall, if large, were joined. Generally it was found that shell wounds of this type showed a far greater amount of muscular damage than was at first apparent. Skin packs were applied to all wounds.

General Abdominal Inspection.—If there were signs of blood loss and the patient was suspected of having a hæmorrhage from a solid organ, the liver and spleen were examined first and the hæmorrhage was controlled. Gut, if damaged, generally underlay the incision as this was made in proximity to the wound. If large bowel was found to be involved and its contents were leaking into the peritoneum, it was delivered—being mobilized if necessary—and the damaged portion was clamped off and covered with a pack. A general inspection of the colon and a complete examination of the small bowel were made and all lesions were dealt with. At this stage a fairly accurate estimate of the course of the missile could be made either from sites of the lesions in the bowel or from evidence of damage at the root of the mesentery or posterior abdominal tissues, and the necessity for further search determined. The stomach was always inspected in wounds of the upper quadrants and in lower chest and flank wounds.

There was nearly always a fairly large quantity of blood in the peritoneum. This had to be removed before a proper inspection could be carried out. A quick and effective method in cases in which a transverse incision was made, was for the surgeon to pull and depress the outer end of the incision whilst the patient was rolled slightly to the side. The remaining fluid was then mopped or sucked out. In all cases in which the intraperitoneal course of the foreign body could not be determined a complete examination of all the abdominal contents was made.

Closure of Wounds and Ligatures.—In transverse incisions very few sutures were necessary for reasonable apposition. It was found that after closure of the peritoneum four or five interrupted stitches, including all muscle layers, were quite adequate. Bowel was always sutured with catgut,

but in the latter stages of the campaign linen thread was used for most ligatures and, as far as can be determined, did not cause any trouble in the healing of the incisions.

Sulphadiazine.—The intraperitoneal use of sulphadiazine in cases of perforated gut was suggested by Lieutenant-Colonel G. A. H. Buttle, Royal Army Medical Corps, Commanding Officer of Number 1 Base Transfusion Unit, and quantities were made available by him for use in these cases. The suggested dose was ten grammes, which was sent up in suspension in 100 cubic centimetres of normal saline solution, ready sterilized in small bottles.

Pre-operative injection of sulphadiazine was considered, but it was felt that there was some risk attached to this procedure, and its use was deferred pending results from injection during or after operation. These appeared to be so satisfactory that its pre-operative use was not thought of again. It would appear to be indicated in the treatment of patients with suspected bowel perforation requiring long evacuation to a surgical team. Its use by the regimental medical officer in jungle warfare seems worthy of consideration.

Technique of Intraperitoneal Injection of Sulphadiazine.—At first an attempt was made to empty the 100 cubic centimetre suspension through the incision into the abdominal cavity. This did not prove altogether satisfactory. A quantity escaped during the suture of the wound and, if there was any straining, most of it was lost. Moreover, it was felt that the greater portion should be in the area of maximum bowel damage and particularly near any large bowel lesion or leak.

The following technique seemed to fulfil these ideas. A small drainage tube, of internal diameter just sufficient to fit easily over a "Vim" syringe nozzle, was introduced to the highest point of the suspected infection, as, for example, at the site of the hepatic flexure in a right colectomy or the site of the highest perforation in wounds of the small intestine. This tube was brought out either through the lateral end of a transverse incision, through a separate stab wound or, in cases of loin wounds, through the original wound if at all convenient and big enough. The abdominal incision was closed and then the suspension was injected. Sometimes a little leaked through the incision, and this may explain the rarity of wound infection despite the fact that the team had no facilities for waterproofing incisions. The tube was clamped off with Spencer Wells forceps for twelve hours. Injection was repeated at twenty-four to thirty-six hour intervals on one or two occasions. If a large pelvic tube had been used as well, it was found necessary to clamp this temporarily during the first injection. A third injection was made only in cases in which there was large bowel soiling or in which a fairly acute peritoneal reaction was noted at the operation.

The following observations were made following this technique:

1. In no case was there purulent discharge from any drainage tube.

2. Release of the Spencer Wells forceps from the sulphadiazine tube was generally followed by a serous discharge for a few hours.

3. No difficulty was experienced in injecting these quantities even seventy-two hours after operation.

4. Drainage tubes, even with lateral holes, left quite undisturbed from four to five days, could be withdrawn easily.

5. In cases in which peritoneal soiling was caused by large bowel contents or contamination of a retroperitoneal hæmatoma was occasioned by an extraperitoneal lesion of the colon, even of twelve hours' duration signs of alarming infection did not appear in these areas after its use.

6. Post-mortem examination of the abdomen following its use showed no evidence of acute peritonitis, an absence of adhesions, a little serous fluid in the pelvis and drainage tubes lying free.

Facilities, unfortunately, were not available for laboratory investigation in these cases. The chart (Figure II), by courtesy of Lieutenant-Colonel Buttle, shows the curve of the blood sulphadiazine following one intraperitoneal injection. It would appear from this that any effect must be a local one, and it is added evidence for a planned injection by the technique described rather than by a blind one.

The routine use of sulphadiazine in bowel perforations was commenced at about the period in which other standard procedures were worked out and adopted by our surgical team. Rapid transfusion and exteriorization of the colon came into practice almost *pari passu* with its use. There is

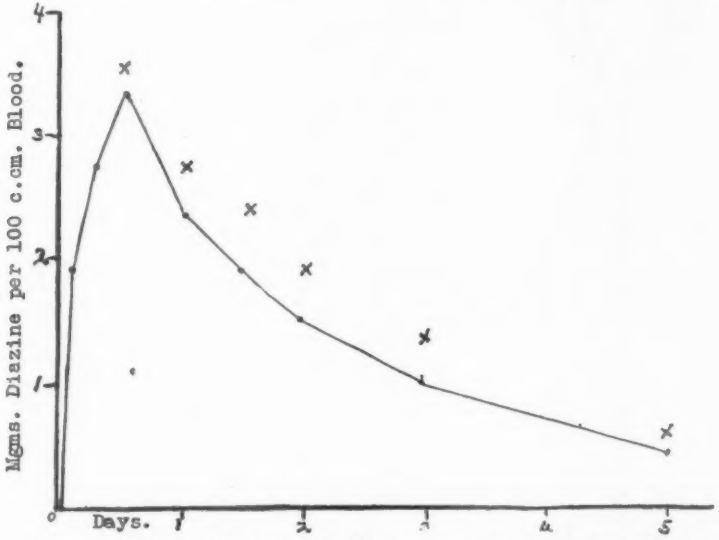

FIGURE II. Chart of sulphadiazine blood curve. Ten grammes of sulphadiazine suspension put into peritoneal cavity at operation for appendicectomy. X indicates total diazine, that is, free acetyl.

therefore difficulty in assessing the part played by these different factors in the improved recovery rate which occurred from this time. But our clinical evidence would suggest that the use of sulphadiazine was by no means an unimportant factor in the reduction of mortality rate, and that it should be used as a routine in all cases of expected peritoneal infection.

The Wound.—Small wounds were not touched. Large lacerations were excised and a routine sulphonamide and "Vaseline" gauze dressing was applied. Where the wound was conveniently placed for peritoneal drainage—for example, posteriorly in the flank—a tube was introduced.

POST-OPERATIVE TREATMENT.

General Nursing and Holding.—As a matter of principle, all patients with abdominal wounds were held for a minimum period of seven days before evacuation to a casualty clearing station. During the Alamein battle some patients with injuries to a solid viscus were evacuated before this, as holding

capacity was strained. And reference will be made later to early evacuation of patients with extraperitoneal lesions of the rectum.

In the early stages of the surgical team's tour of duty, patients, of necessity, were nursed on stretchers. Patients with abdominal wounds were all placed in Fowler's or semi-Fowler's position, which, even with air cushions and folded blankets under the knees, was far from comfortable. No very satisfactory backrest could be fitted to a stretcher. In September, eight hospital beds were issued, and a further seven and backrests for the whole fifteen were made by a field park company. In these the patient's convalescence was much smoother and more comfortable.

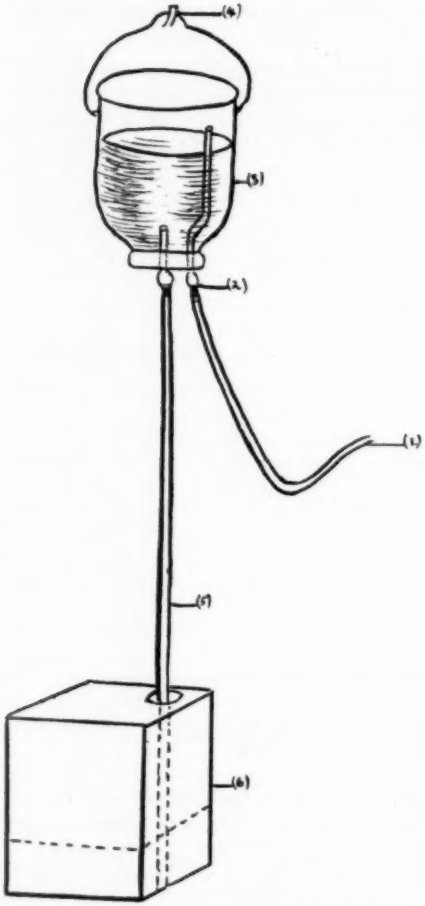

FIGURE III. "Soluvac" for gastric suction. (1) = glass connexion to gastric tube; (2) = tied with linen thread to end of glass tube in "Soluvac"; (3) = "Soluvac" flask; (4) = suspended from tent roof or from stand; (5) = tube under water; (6) = petrol tin on floor.

Gastric Suction. — The post-operative nursing proved a problem only in patients with an injury to a hollow viscus. Most were given fluid intravenously, mainly glucose saline solution. At first there was no routine gastric suction for patients who had bowel lesions, and one after another of the patients developed ileus. The only tube for suction at our disposal was a large stomach tube. A request was made for Ryle's tubes and finally they arrived. These were found by no means satisfactory, for as nursing was carried out by only a few orderlies, they could not receive constant attention and were continually becoming blocked. Finally rubber drainage tube about three-sixteenths of an inch external diameter and sufficiently firm not to kink in the naso-pharynx or to collapse with suction was procured and home-made suction tubes were designed, pieces of lead or solder being tied into the end and fairly large lateral holes cut. These gave no trouble and required a minimum of supervision. Captain Berryman suggested the use of "Soluvac" bottles for continuous gastric suction (see Figure III), and the set-up as illustrated worked admirably. At first suction was started only when the clinical picture of ileus—the rise in pulse rate, the greying colour of the patient, and the increasing distension of the abdomen—had developed. Suction immediately improved the general appearance and the pulse rate dropped, but many of these patients still did not recover. Finally, as ileus appeared to occur in practically all cases, it was decided to anticipate it and suction was started as soon after operation as the patient could swallow the

tube. With the adoption of this technique no further patient developed ileus and much anxiety in cases of bowel suture or resection disappeared. The mortality rate in these cases dropped considerably.

Suction was always maintained until bowel sounds could be heard by auscultation. This was first listened for on the third post-operative day. The suction was then discontinued for several hours, with the tube still remaining in the stomach. At the end of this time some patients still had distension. In these cases suction was again started, continued for another twenty-four hours, and then disconnected once more. If no discomfort was felt, the stomach was emptied and the amount of fluid measured. If this amounted to only three or four ounces, the tube was removed. If the quantity was larger, suction was resumed for a further twenty-four hours and then the whole process was repeated. Most patients felt so much more comfortable with a tube in that they were loath to give it up. They knew that vomiting and even nausea disappeared immediately suction was commenced.

Post-Operative Intravenous Administration of Fluids.—Patients who were subjected to gastric suction needed continuous transfusion of fluids. Amounts given were: 3,500 to 4,000 cubic centimetres for the first twenty-four hours and then 3,000 to 3,500 cubic centimetres until 1,000 cubic centimetres of urine were excreted in twenty-four hours. When this excretion was occurring a balance was struck, the quantity of urine, of aspirated gastric content, and 1,000 cubic centimetres for invisible loss being placed on the debit side, and the amount of intravenous fluids given on the credit. A positive balance was always maintained. Orderlies quickly learned how to work out fluid balance charts. At first the only solution available for use was a 5% glucose in normal saline solution, but with this œdema due to chloride retention developed after a few days. Captain F. Connaughton then suggested having two solutions made up, one a 5% glucose and the other normal saline solution. From these, reasonable chloride requirements could be met and the balance of the fluid needed given as 5% glucose solution. Chloride requirements were estimated according to standards laid down by Naunton Morgan,[1] namely, a basic ration of five grammes or 600 cubic centimetres of normal saline solution per day *plus* replacement of all fluid removed from the stomach. If the abdomen was distended, a further 600 to 1,000 cubic centimetres were given to compensate for chloride loss in the bowel. The balance of fluid administered was 5% glucose solution. Number 1 Base Transfusion Unit readily cooperated and put up the solutions separately. From the time of commencing this technique no further patients developed œdema or became water-logged, although some were given fluids intravenously for seven days.

Sulphonamide 5% in glucose solution was also available and was used when sulphadiazine was not given. Two or three bottles of 500 cubic centimetres each were given over a period of twenty-four hours. There were no facilities for estimating plasma albumin and globulin, but after the third day, in cases in which continued intravenous therapy was required, 600 cubic centimetres of wet serum were generally given every twenty-four hours with apparently very definite improvement.

NEW STANDARDS SET FOR ABDOMINAL CASUALTIES.

Thus, as experience grew, it was found that, in the conditions under which the team worked, certain standards could be set for the treatment of abdominal casualties in forward areas. In mobile warfare the standards would not be impossible, although some of them would be harder to attain. In these circumstances it would be necessary to have posts at which patients with abdominal wounds were left under the care of a medical officer and one or

two trained orderlies until they were fit to be evacuated. Emphasis must be laid on the fact that unless the surgeon can be sure that his abdominal patients will not be moved during the first seven post-operative days, he should not operate on them. Movement—even of a mile or so—during this period is most dangerous. It has been proved beyond any doubt that there is less risk in evacuating the patient and thus deferring operation for some hours than there is in performing operation earlier and evacuating the patient during this dangerous post-operative period. These set standards may be classified as follows.

A. *Pre-Operative.*

1. Reduction of time-lag—the time between wound and operation. This can sometimes be reduced by moving forward a main dressing station and associated surgical team; but this lag can always be kept at a minimum in regard to the time spent in resuscitation at the main dressing station by rapid transfusion.
2. Reduction of operation risk. This can be brought about by presenting for operation a patient who, however severe his injuries may be, is still a good operative and anæsthetic risk—that is, he has a systolic blood pressure stabilized at 100 millimetres of mercury or over and a reasonable colour.

B. *Operative.*

1. Maintenance of patient's general condition despite a major operative procedure—that is, maintenance of his condition if necessary by rapid transfusion during operation.
2. The use of sulphadiazine in all cases of perforated small or large bowel.
3. The employment of safe methods of colon surgery (see "colon injuries").

C. *Post-Operative.*

1. Early and continuous gastric suction in all cases of perforated bowel.
2. Continuous intravenous therapy for some days, and the proper regulation of this so as to maintain a positive balance and avoid œdema.
3. Comfort and rest during this period by nursing the patients in proper beds.

These were the standards attained by our surgical team during the last half of its tour—called in the forthcoming table Phase 2. During this period all patients, with the exception of two prisoners of war found some twenty-four hours after being wounded, were ready for operation in under twelve hours from time of being wounded. A great improvement in results—particularly in wounds of the small and large intestines—followed the adoption of these standards.

RECOVERY RATE.

It was found possible to follow up over 95% of all patients (see Table I) and so a recovery rate—as distinct from number of patients evacuated to base—can be shown. "Recovery" is arbitrarily a live patient one month after operation. Case CXXVI—with death in the fifth week—is, as far as can be determined, the only fallacy in this table.

TABLE I.

Showing the Recovery Rate, in Two Phases, of all Patients, 76 in Number, with Intraperitoneal Injury.

Phase.	Number of Cases.	Number Recovered.	Recovery Rate.
1	33	14	42·0%
2	43	31	72·0%

If the 43 cases of perforation of small bowel and/or colon are divided into the two phases, an even more striking improvement is to be noted (Table II). It is in these that effects of gastric suction, post-operative intravenous therapy

TABLE II.

Phase.	Number of Cases.	Number Recovered.	Recovery Rate.
1	18	5	28%
2	25	18	72%

and sulphadiazine are shown to the full, for every one of these patients in Phase 2 was so treated.

In all its activities our surgical team was most fortunate in having the interest and cooperation of the medical services, not only of Australian Division and 30 Corps, but also those of General Headquarters, Middle East. It was in great part due to this, and to the prompt dispatch of everything requested, that the above standards were made possible and the consequent improvement in results was attained. Particular reference must be made to Number 1 Base Transfusion Unit, which maintained, even in rush periods, a most complete and adequate supply of all fluids for intravenous therapy, as well as blood and serum. Frequent visits by members of this unit to forward areas enabled them to appreciate the problems and to afford the team the benefit of their helpful suggestions.

CONSIDERATION OF SPECIAL LESIONS.

WOUNDS OF ABDOMINAL WALL WITH NO INTRAPERITONEAL DAMAGE.

A. *Laparotomy: No Peritoneal Involvement* (nine cases).—In five patients wounds were present in the anterior abdominal wall and associated with these was an abdominal rigidity. Large wounds were excised and followed down to the peritoneum. This was opened widely enough for the surgeon to be able to evert and inspect the parietal peritoneum for some distance beyond the wound track. Most of these five patients showed hæmorrhage and bruising in a plane just superficial to the peritoneum. In a few injection of the peritoneum was present. Four patients had wounds in the buttock. Three of these had abdominal rigidity. The fourth patient showed a greatly diminished liver dulness, and exploration would not have been carried out but for this observation. A very small right lobe of the liver was found, scarcely larger than the normal left lobe.

B. *Laparotomy: Perforated Peritoneum as only Abdominal Lesion* (two cases).—One patient had a wound of the right side of the lower part of the chest, fracturing the ninth and tenth ribs and leaving a peritoneal defect of nearly one inch in circumference. Laparotomy proved that the wound was tangential and that no intraperitoneal damage had occurred. The patient died of pneumonia following evacuation to a casualty clearing station. The other had blood in the peritoneal cavity arising from a diaphragmatic perforation. On his left side was a hæmothorax, which was the source of the blood.

C. *Retroperitoneal Hæmatoma.*—Whilst the anterior and lateral abdominal walls are comparatively thin, contain no major vessels and are completely covered by parietal peritoneum, a very different state of affairs exists in the posterior wall. Here on a thick muscular wall, deeply situated, lie main vessels with numerous branches, and on either side the fixed portions of the colon in direct contact with the retroperitoneal tissues.

Wounds of the flank and loin, when caused by medium or low velocity projectiles, are less liable to penetrate the peritoneal cavity because of the thickness of this muscular layer. Trauma in the region of the vascular layer,

however, may produce a large hæmatoma, which is most commonly seen beneath or medial to the fixed portions of the colon. If the larger vessels are cut the hæmatoma may spread over the whole of one side of the abdomen, from the renal area down over the brim of the pelvis to the bottom of the rectovesical space; indeed, the true pelvis may be completely filled by it. More commonly a smaller hæmatoma is found, floating up the fixed part of the colon of that side and extending inwards as far as the great vessels.

Reports were current in the Middle East that in some hospitals these retroperitoneal hæmatomata were associated with a high mortality, that patients died from the seventh to tenth day of a spreading cellulitis in the retroperitoneal tissues, and that this had its origin from a hæmatoma

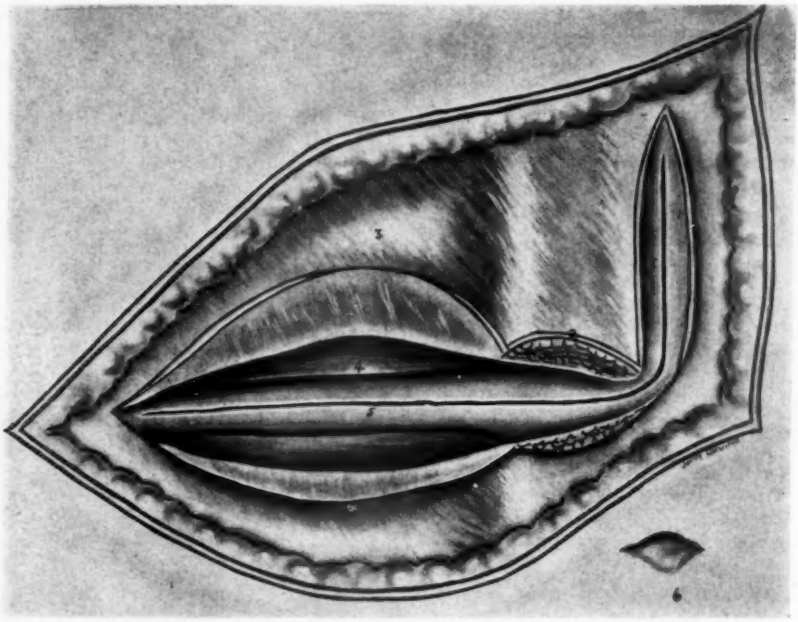

FIGURE IV. Transverse abdominal incision with prolongation upwards. 1 = prolongation of the incision in the mid-line; 2 = rectus muscle and sheath cut across; 3 = external oblique muscle; 4 = internal oblique muscle; 5 = peritoneum with incision; 6 = umbilicus.

secondarily infected from an overlying colon. In view of these reports it was decided that in all cases in which a hæmatoma of reasonable size was found—that is, where it actually lifted up the fixed colon—it would be adequately drained. As in the majority of the cases in this series there was the doubt of involvement of ascending or descending colon, exploration was carried out in most of them through a transverse incision (see Figure IV). If no intraperitoneal lesion was found, extraperitoneal tearing of the colon could not be excluded. In these circumstances an extension of the transverse cut was made (if necessary), and then with the finger the paracolic peritoneum was stripped from the abdominal wall till the hæmatoma was finally entered (see Figures V and VI). When involvement of the colon was present a fæcal smell was generally the first indication of such a lesion. Palpation of the extraperitoneal surface of the fixed colon would then reveal the perforation. The colon was then fully mobilized and dealt with (see "colon injuries").

If there was no smell and palpation of the overlying colon revealed no hole, it was assumed that only a hæmatoma had to be dealt with. In this case the original wound, if it was large enough, was used for drainage; if it was not, drainage tubes were introduced extraperitoneally through the lateral end of the transverse incision or through a stab in the flank. Hæmatomata which extended into the true pelvis and lay in contact with the extraperitoneal surface of the rectum or bladder were drained by an additional tube.

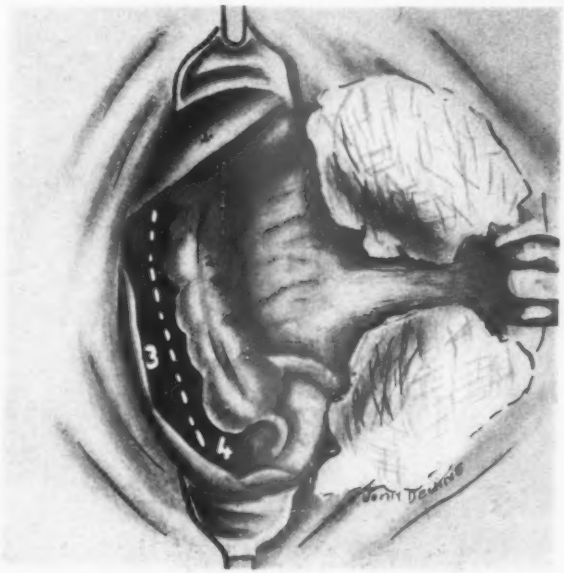

FIGURE V. Showing incision in the peritoneum and the paracolic gutter. 1 = liver; 2 = ascending colon; 3 = incision in paracolic gutter; 4 = parietal peritoneum.

Seven cases of medium or large hæmatomata, not associated with a bowel lesion (in four of these the mesentery was involved), were dealt with in this series. All the patients recovered after fairly free drainage for three or four days, when the tube was removed. In these cases there was no evidence of cellulitis, nor was there any anxiety over the patients' convalescence.

Whilst not typical of retroperitoneal hæmatoma, one case—Case CLIX—(see appendix) is of interest. Although for statistical purposes this case was grouped under "stomach and small bowel injuries", death was thought to be caused by a secondary vascular rupture, possibly of the aorta. Unfortunately, as the patient died during a period of continuous work, an autopsy could not be made.

WOUNDS WITH INTRAPERITONEAL DAMAGE.

There were 76 cases in which abdominal wounds were accompanied by intraperitoneal damage.

Liver and Gall-Bladder.

There were 14 cases in which the liver and gall-bladder were involved and 12 of the patients recovered (see Table III).

TABLE III.

Site of Damage.	Number of Cases.	Remarks.
Liver only	4	——
Liver and gall-bladder ..	1	Case CXXV.
Liver and bowel ..	4	Two deaths: (1) Large tear in splenic flexure eighteen hours old. (2) Case CLVII. One case of gall-bladder involvement was included in this group (Case CXVII).
Liver and kidney ..	2	Nephrectomy required in one.
Liver and thorax ..	3	One required suture of diaphragm (Case CXXIV).

Four of the above patients had other severe injuries—namely, major compound fractures; but so many patients with abdominal wounds had such associated injuries that it has been found impossible in any classification to make any reference to them, and they have been ignored except where they were thought to be a probable cause of death.

Treatment.—In nine of these cases, at operation, hæmorrhage was found practically to have ceased. In these nothing was done to the liver wound.

FIGURE VI. Mobilization of caecum and ascending colon. 1 = edge of parietal peritoneum from incision in paracolic gutter; 2 = shrapnel wounds in the retroperitoneal part of the ascending colon surrounded by hæmatoma and ecchymosis; 3 = posterior surface of peritoneal leaf infiltrated with blood; 4 = muscles of the posterior abdominal wall infiltrated with blood from which the ascending colon has been turned forward; 5 = small intestine with mobilized ileum with adjacent appendix; 6 = duodenum; 7 = kidney from which merges the ureter which is later joined by the genito-crural vessels.

Three patients had large ragged lacerations, still bleeding. All these lacerations were on the superior or lateral surface of the right lobe. Hæmorrhage was controlled by packing with gauze. This was removed on the third or fourth day under "Pentothal" anæsthesia. In the case of one man with a large shell wound in the posterior axillary line through the lower ribs and a tear high up on the lateral surface nearly large enough to take a fist, packing was introduced through the original wound—that is, transpleurally. Subsequently, at a base hospital, he developed an infected hæmothorax. In the others packing was brought out subcostally through the lateral end of a

transverse incision. In two cases the lacerations were sutured. A free omental graft was laid over the tear and through this the stitches (which held well) were introduced. In all cases in which packing was used or suturing was carried out, Morrison's pouch was drained.

Gall-Bladder.—In two cases (Cases CXVII and CXXV) the gall-bladder was involved as well as the liver. In each the wound was caused by a relatively small shell fragment passing through the liver and the gall-bladder within an inch of its fundus. In each case generalized board-like rigidity was an outstanding feature. In one the peritoneal cavity was almost filled with frothy bile-stained fluid. In the other there were coincident perforations of the transverse colon and of the jejunum; and there was little evidence of bile in the peritoneal cavity. Cholecystostomy and drainage of Morrison's pouch were carried out in each case. Both patients recovered.

Spleen.

There were three cases of damage to the spleen and two recoveries. All three were uncomplicated by any other visceral lesions.

The two patients who recovered, despite a fairly large quantity of blood in the peritoneum and the need for transfusion, were found to have only small wounds in the spleen. In one the wound was sutured over a free omental graft: in the other nothing was done, except closure of the entry wound in the lower part of the chest, which had commenced to suck.

In the fatal case (Case XXXI) transfusion was given slowly, and after 2,500 cubic centimetres of blood had been transfused the patient's condition had shown little improvement; he was still a poor risk. At operation the splenic pedicle was found to be severed and splenectomy was performed. Death occurred in twenty-four hours. The post-mortem examination showed no further hæmorrhage and no overlooked injuries. Prolonged severe shock was assumed to have caused irreparable damage to vital centres.

Rapid transfusion would have probably prevented this and saved the man.

Kidney.

There were ten cases of kidney damage, and among these there were six recoveries (see Table IV).

TABLE IV.

Lesions Found.	Number of Cases.	Remarks.
Kidney only	1	Transected pedicle. Nephrectomy. Death. (Case VII.)
Kidney and liver ..	2	Nephrectomy in one.
Kidney and stomach ..	2	Both late; done in Phase 1; nothing done to kidney. Both patients died.
Kidney and colon ..	4	All patients recovered; three nephrectomies. (Cases III, LXXV, XCV, CXLIII.)
Kidney and multiple abdominal injuries ..	1	Irreparable damage; death. (Case CLVII.)

Significant points to be noted from this table are: (i) In nine out of the ten cases other visceral damage was present. (ii) In half the cases the colon was involved; only one of the patients (Case CLVII), whose colon was involved, died.

Analysis of Deaths.—Apart from Case CLVII the other three deaths occurred in patients operated on during the Phase 1 period. The two patients in whom the stomach was involved were both submitted to operation late (over twelve hours), as bad risks, after prolonged "slow resuscitation". Both had large perforations of the stomach which were closed, but nothing was

done to the kidneys. Similar slow, ineffective resuscitation was given in the only case of uncomplicated renal damage. Rapid nephrectomy in this almost pulseless patient revealed a tear extending into the renal pedicle. This man would have been certainly saved with "rapid transfusion" and early operation, and might have been saved if operation, as a bad risk, had been undertaken some hours sooner.

It is felt too that the two men with complicating stomach injury could have been made "good risks" speedily if they had been given rapid transfusions, and one at least would have been submitted to operation in the six to twelve hour period.

Hæmaturia.—The urine of all patients with wounds that might have involved the kidneys was examined before operation so that the incision could be planned to give access to the kidney. If necessary, their bladders were catheterized.

Management.—Laparotomy was performed in all but Case VII. In this patient a routine nephrectomy was performed. The incision was transverse or subcostal in all but one. This gave an excellent approach to the kidney, colon and contents of the upper part of the peritoneal cavity.

Of the six patients who recovered, nephrectomy was done for four; one (Case III) had a retroperitoneal hæmatoma drained, and one, with liver injury as a complication, was treated conservatively.

It is considered that in wounds of the kidney complicated by damage to the colon there is no indication for conservative surgery. Particularly is this the case if there is a perirenal hæmatoma, which is nearly always present. In these circumstances infection in the perirenal tissues is almost certain to occur and this is likely to spread to the damaged kidney. And a secondary nephrectomy is a more difficult procedure and attended with a higher mortality.

In this series, of the four cases associated with colon damage, nephrectomy was performed in three, and in two of these resection of half the colon was also necessary; nevertheless the convalescence of all the patients caused little anxiety.

In Case III the patient, by recovering, gave a false picture of the dangers attendant on colon damage, and delayed the application of "exteriorization tactics" (subsequently described) in the surgery of this region.

In all cases drainage was carried out from the flank, either through the original wound or the lateral end of the transverse incision. In Phase 2 sulphadiazine was used in the treatment of all patients who had associated bowel injury.

Bladder.

There were five cases of bladder injury, with three recoveries. The two deaths occurred in Phase 1. The cases may be divided into two groups:

Group A: The bladder alone was involved in two cases. There was one death.

Group B: Injury to the bladder was associated with some other visceral injury in three cases. In this group there was one death.

In four of the cases there was an intraperitoneal perforation of the bladder. All the patients had extraperitoneal damage.

The two cases in Group A were, to a point, very similar. Both patients had extensive tangential wounds of the anterior abdominal wall, fracturing the symphysis, with a blood-stained urinous discharge. Both were late cases, the patients being received over twenty-four hours from wounding. There was, however, one important difference.

In one there was no abdominal rigidity or marked tenderness and it was reasonably certain that there was no peritoneal involvement. The wound was excised, loose pieces of pubis were removed and a large anterior bladder defect was closed round a large tube, and the peritoneum, which appeared intact, was not opened. This patient progressed satisfactorily.

The other, whose general condition was poorer, had great rigidity and commencing distension of the abdomen. The peritoneal cavity was opened and found to contain a moderate amount of stale blood-stained fluid in the pelvis. Injury to the bladder had involved the peritoneal coat for a short distance. A similar suprapubic cystostomy was performed and the peritoneum was drained. The patient died in twenty-four hours.

In Group B visceral damage was limited to the small intestine. The position of the wounds was of some interest.

In the only fatal case the patient had a penetrating shell wound of the perineum just lateral to the urethra, but not damaging it, and perforations of jejunum and ileum.

In one case (Case CXXVII) the entrance wound was just below the left posterior superior iliac spine, the exit wound just above the symphysis. In its course the bullet had perforated jejunum and ileum (multiple wounds in each) and the pelvic mesocolon and, in addition to perforating the bladder, caused a lateral hole of the left internal iliac vein.

In the third case (Case CXXXIV) the foreign body, also perforating, entered the left buttock and made its exit in the right thigh just below the anterior superior iliac spine. One large tangential tear of the lower ileum was the only intraperitoneal damage.

In the fatal case the man would probably have been saved with immediate post-operative gastric suction and sulphadiazine. By the time suction was started he was irrational and the tube could not be kept in position. Cases CXXVII and CXXXIV, both of which were treated during the Phase 2 period, show in marked contrast the value of the standards set for the post-operative treatment of this period.

Methods of Treatment.—Intraperitoneal lesions were repaired and suprapubic cystostomy was performed. Extraperitoneal damage of the bladder was left alone except, where there was a defect in the anterior wall, to close this round a large suprapubic tube. After evacuation of hæmatomata, prevesical and paravesical spaces were widely drained, if necessary, on each side.

Stomach.

There were seven cases of stomach injury with one recovery (see Table V).

TABLE V.

Site of Lesions Found.	Number of Cases.	Remarks.
Stomach only	1	——
Stomach and bowel	1	Case CLIX—? aortic rupture.
Stomach and kidney	2	Both late.
Stomach and liver	1	Only recovery—Case CXXXV.
Stomach and thorax	1	Case CXXXVIII.
Stomach and multiple abdominal wounds	1	Case CLVII.

The following facts to some extent explain these very unsatisfactory figures in Table V.

In five cases the wound was caused by large shell fragments, and perforations of both walls of the stomach the size of a florin or larger were found. In three of these operation was not performed until after twelve hours from the time of the wounding. With rapid transfusion one of the three patients would have been operated on hours earlier and would have had a much better chance of recovery. Of the remaining two, one (Case CLVII) had irreparable damage; the other patient (Case CXXXVIII) had a severe

abdomino-thoracic wound the details of which are cited in the appendix. This latter patient died of an empyema after his evacuation to base.

Of the two patients with small gastric wounds, one (Case CLIX) has already been mentioned as having died from sudden aortic rupture. The remaining patient (Case CXXXV), the only one who recovered, is mentioned to emphasize the importance of laparotomy in any doubtful perforating wound. In his case operation was nearly not performed because there was no rigidity and very little tenderness in the neighbourhood of the wound in the right epigastrium.

Sulphadiazine was not used in the treatment of any stomach wounds as it was felt that the spilled contents were not sufficiently infected to warrant its use.

Small Intestine.

There were 28 cases of injury to the small intestine, with 13 recoveries (see Table VI).

TABLE VI.

Lesion Found.	Phase 1.		Phase 2.	
	Cases.	Recoveries.	Cases.	Recoveries.
Small intestine only	4	1	4	3
Small intestine and colon	2	0	9	7
Small intestine and other hollow viscera ..	2	0	3	2
Small intestine and other injuries of a gross nature	1	0	3	0
Total	9	1	19	12

In Phase 1 most of the patients operated on for injuries of the small intestine died in ileus; and even if all the complicated cases are excluded, a thoroughly unsatisfactory recovery rate of 25% is found. But, as will be seen in Table VI, patients operated on in Phase 2 for a similar type of injury show a remarkable contrast to this: in 19 cases there were 12 recoveries.

Other gross injuries were a certain cause of death in two of the three cases in this phase.

In one case, when a post-mortem examination was made, the bowel perforation was found to be completely healed with no signs of peritonitis. In this case one leg had been amputated for suppurative myositis at a secondary operation. At the autopsy this condition was found to be widespread throughout the other leg.

In the second case, with a shattering shell wound of the right hip, a ligation of the right common iliac artery was found necessary to control bleeding from most of the branches of the internal iliac and deep femoral arteries of this side. Before operation this man required a transfusion of 1,800 cubic centimetres of serum and 8,500 cubic centimetres of blood. The artery was approached by the transperitoneal route and, as a complication to the patient's other very severe injuries, two small perforations of the ileum were found; these were closed. He died in eighteen hours.

The third patient, a prisoner of war, came for treatment thirty-six hours after wounding. The small bowel was prolapsed and the muscles of the anterior abdominal wall and left thigh were dark and crepitating. It was assumed that the injured muscles were the seat of gas gangrene; and after resection of the bowel the abdominal muscles were not sutured. The patient died before it could be ascertained whether the wound would burst open if the bowel were kept from distending. (Case CXLIX.)

Complicating injuries in "other hollow viscera" were injuries to the bladder in two cases, both with recovery (Cases CXXVII and CXXXIV), and injuries to the stomach in one case (Case CLIX). To all these reference has been made previously.

Excluding the deaths from "gross other injuries", Table VI shows the very satisfactory figure of 75% recovery rate in the surgery of the small intestine during Phase 2.

Division of the cases into lesions of the jejunum and those of the ileum shows little difference in mortality in these two regions of the gut; but, as in over 25% of cases both regions were involved, the numbers are rather small for definite conclusions to be drawn from them.

Coincident injury of the colon does not appear to raise the mortality. This is to be noted particularly in Phase 2 period, during which an added and new operative factor entered the picture—namely, "exteriorization" of all colon injuries. On this factor stress will be laid in the discussion of colon injuries.

Duodenum.—There were two cases of duodenal injury (Cases CXX and CXXXIII). Both were extraperitoneal and complicated by other bowel lesions. In Case CXX the patient died suddenly on the operating table of respiratory failure. In Case CXXXIII, with two duodenal perforations and multiple jejunal perforations, the patient did well without drainage of the retroperitoneal space. Some six weeks after operation he was seen at an Australian general hospital. At that time colicky pains and some vomiting after food were noted, and it was felt that this might be due to a stenosis of the duodenum. The symptoms, however, disappeared after four weeks.

Clinical Observations on Wounds of Small Intestine.

Certain points were at first striking to one unaccustomed to operating on abdominal wall wounds.

Blood Loss.—Blood loss was surprisingly large, even in patients who did not appear greatly exsanguinated; and it would seem to be quite a major factor in the production of their shock. In practically all cases the peritoneal cavity was fairly well filled with what seemed to be almost pure blood. This has been noted by other observers.[2] The source of this hæmorrhage was, in general, the lacerated bowel. Bleeding was more marked in jejunal than in ileal lesions.

Multiple Perforations.—Multiple perforations were often a great distance apart. For example, one might be in the upper part of the jejunum and the other in the lower part of the ileum. This is not surprising when one considers the disposition of the gut in which early jejunal coils lie in the left iliac fossa. It is strikingly brought home when one makes a routine examination of the whole bowel.

What might be termed a "jejuno-colic lesion" was found on several occasions associated with wounds in the left iliac fossa or left loin. In this lesion injury to the lower descending colon or pelvic colon or mesosigmoid was found with an injury to the jejunum some eighteen inches from the duodeno-jejunal junction. After the discovery of this syndrome, when, in wounds in the above-mentioned regions, a lesion of the pelvic colon was found, the next step taken was to make a complete examination of the small intestine, starting from the upper end and working down. Case CLI illustrates this type of jejuno-colic injury.

Gross Injury of the Mesentery.—It was expected that not a few casualties would show tearing of the mesentery, with devitalization of gut which would necessitate resection. These mesenteric lesions were unexpectedly absent. The only "uncomplicated" mesenteric lesion, not included in the 28 cases analysed above, was that caused by a small shell splinter, when a mesenteric vessel required ligation.

In five cases a resection was necessary, in every one for local damage to the bowel. In two of them this damage extended over many feet.

Prolapsed Bowel.—All patients with prolapsed bowel who were dealt with, died.

Operative Treatment in Injuries of Small Intestine.

Closure of Perforations in the Small Intestine.—In 23 cases perforations in the small intestine were closed; there were 12 recoveries (16 of these patients were in Phase 2, and among these there were 11 recoveries).

Resection of Damaged Segment.—The damaged segment was resected in five cases; one patient recovered.

Resection.—The greater mortality rate which resulted from resection was recognized, and perforations were sutured however many or large they were, unless a portion of the bowel was damaged irreparably or these repairs would cause dangerous kinking or a stenosis. Catgut sutures in two layers were used. The mortality following resection is believed to be due to the seriousness of the initial injury and to the consequent shock. Any measures that will lessen the time of operation and the risk of peritoneal infection should, therefore, reduce this mortality. In this respect the author feels that this could be accomplished by a resection of a small gut "extraperitoneally" (with an ileostomy, or jejunostomy, and a spur)—that is, by an operation comparable to an ileo-colectomy done on the Paul-Mikulicz principle with a spur. In these circumstances, in order to avoid unnecessary loss of intestinal contents, the spur should be crushed within a few days. One patient (Case CXXVI) was treated on this principle, and lived for five weeks following resection of nearly two-thirds of the small bowel. Death was due to an infective phlebitis and a secondary endocarditis. The smooth post-operative course of this patient, with his ileostomy and colostomy, was remarkable. After three days his gastric suction was discontinued when his ileostomy immediately functioned. At this time no enterotomes were available for dealing with the spur, and the spur had to be left uncrushed for far too long a time.

This operation should prove of particular value in cases in which closure of the abdominal wound is considered dangerous—for example, in Case CXLIX, to which previous reference has been made. With suction and with ileostomy there should be little risk of the bursting open of the abdominal wall.

Post-Operative Care.

Mention has been made on several occasions of continuous gastric suction and the intravenous use of fluids. It is in small bowel lesions that the routine and early post-operative use of this method in all cases produces its most dramatic results. During the Phase 2 period of abdominal surgery no case of ileus or post-operative vomiting occurred after suction had been started. Gastric suction should always be continued until peristalsis is well established.

Colon.

There were 26 cases of injury to the colon, and among these 16 recoveries (see Table VII).

The Evolution of an Operative Policy in Wounds of the Colon.

The great improvement to be noted in Phase 2 was due, in addition to the factors mentioned under the heading of general management, to a very radical change in operative procedure—namely, to a rigid following of the principles of exteriorization of the injured part of the colon. This principle of operation was adopted just about the time when Phase 2 period started.

TABLE VII.

Lesions Found.	Phase 1.		Phase 2.	
	Cases.	Recoveries.	Cases.	Recoveries.
Colon only	8	3	3	2
Colon and small intestine	2	0	9	7
Colon and kidney	1	1	3	3
Total	11	4	15	12

A description of the evolution of this method of operating which was brought about by the surgical team, may be of some interest and may prevent the initial mistakes of the team from being made by others.

Principles of safer colon surgery, laid down by Devine,[3][4] in which ileo-colectomy was carried out by the Paul-Mikulicz principle and a four-inch spur, and in which operation is performed on a defunctioned colon, had been followed in civil practice, but the latter could hardly be applied in urgent war surgery of wounds of this portion of the gut.

Case III was the first abdominal operation of the team's tour of duty. This case did much to hinder the development of principles of safety of colon surgery. In this two small intraperitoneal perforations of the hepatic flexure were closed, and the patient lived.

The next four patients had perforations of the colon. These were closed and all of the patients died. The cases, however, were late; and, in two of them, the repair of the colon was associated with partial resection of the small intestine. All of these patients died in ileus, and died at a time when the problem of dealing with ileus by gastric suction was occupying our minds, perhaps to the exclusion of thoughts about any weakness in operative technique.

Then came the heartening and surprising result in Case XXXVIII.

The patient was a man with a traumatic colostomy that had been left as being too late to justify operation while other injured were waiting for attention. This patient maintained his condition and after twenty-two hours stood a large bowel resection well and, what was more important, had a comparatively smooth convalescence and needed no gastric suction.

It was then decided that in no other injury of the colon would perforation be sutured without a colostomy of the damaged loop or one proximal to it which would act as a "safety valve".

The next two patients were both seen in under six hours after wounding and had small perforations of the colon: one was a perforation of the transverse and the other a perforation of the pelvic colon. These were closed and the patients did well. The importance of keeping all damaged sections of colon outside the abdomen was temporarily forgotten.

Then in the next colon injury (Case LIII) principles were misapplied.

The patient had several small perforations in a fixed portion of the colon—the splenic flexure. A limited mobilization of this segment was carried out, sufficient just to bring it to the surface, and the perforations were closed. There was a rent in the mesocolon, but the gut appeared quite viable. Sulphadiazine too had just come to hand. Instead of more adequately mobilizing the colon, making a spur between descending and transverse colon, and leaving the damaged part as a colostomy, an attempt to "defunction" the distal colon during healing was made by performing a transverse colostomy with spur. The patient died on the fifth day. Although at post-mortem examination there was a collapsed left lung, there was also this important finding: there was a devitalized segment of the colon in the splenic angle.

This observation impressed on us the intrinsically poor blood supply of the colon, and the fact that any impairment of circulation found at operation is certain to be worse after a repair operation has been performed and a reaction has supervened.

A definite policy could now be laid down for all traumatic colon surgery. If the damage was strictly localized, and especially if it was in free portions of the colon, colostomy of the affected loop should be performed; if the damage was in the fixed portions, and particularly if the damage was more than a through-and-through perforation, or if in addition any mesentery was damaged, a formal resection of that half of the fixed colon should be done. These were the principles of "exteriorization" as advocated by Ogilvie,[5] and, conforming to these, no surgeon might repair or suture a colon injury and return it to the abdominal cavity.

The acceptance of these principles for future colon surgery almost coincided with Phase 2 of the team's cases and so to a large extent the improved mortality rate in this period is the result not only of improved methods of resuscitation and post-operative care but also of the adoption of an operative procedure based on sound principles.

These principles were broken only once after this, and that was for special reasons.

In Case CXII a very fixed and adherent caecum (post-appendicectomy) had its lower portion shot away. Rather than tediously free all adhesions, the tear was closed and the suture line stitched extraperitoneally. Convalescence was not uneventful. Complications included an infected wound, a fæcal fistula, and also a late secondary hæmorrhage of the inferior epigastric artery.

Exteriorization in colon wounds, we feel, takes a very comparable place to "excision" of wounds in limbs or in soft tissues; the return of a repaired colon to the abdomen is no more justifiable than is the primary suture of a wound of the soft tissue. This "primary suture" may be successful—as in Case XLVI—but an unnecessary risk has been taken.

Table VIII shows types of operations performed and the improvements in results following the principles of exteriorization.

TABLE VIII.

Operation.	Phase 1.	Phase 2.
No operation	1 (Case XXX)	—
Suture of perforation	7 (4 deaths)	1
Suture *plus* proximal colostomy ..	1 (1 death)	—
Colostomy of affected loop	—	9 (1 death)
Resection	2 (1 death)	5 (2 deaths)

Operative Technique.

Colostomy.—Colostomy was always done with a four-inch spur—that is, afferent and efferent loops sutured together for this distance. Glass rods were unobtainable, so rubber tubes were used to anchor the colon. No sutures were used between colon and peritoneum or abdominal wall. If there were large rents in the affected colon, the colostomy was trimmed at operation. If there were only small perforations with little leaking, it was left for twenty-four hours and then opened widely.

Left inguinal colostomy was done through a separate incision or the outer end of the long oblique incision used for exploration (see Case CXXVI).

Transverse colostomy was performed through the median or paramedian incision or through the medial end of a transverse incision (see Case CXVII).

Two colostomies of the descending colon were necessary (Cases CXLIII, CLXVII). In each case access was gained through a transverse incision allowing a free mobilization of the splenic flexure necessary for the requisite length of spur.

Partial Colectomy.—Partial colectomy was found necessary only in injuries of the fixed portions of the colon. All of these operations were done through a transverse incision. Not only was it found that wounds of these portions of the colon were more extensive, particularly in the case of extraperitoneal lesions, and were associated with retroperitoneal hæmatomata, but also viability of the gut (see Case LIII) was doubtful. So, for these reasons, free mobilization of these fixed parts of the colon was always carried out: on the right side from hepatic flexure to lower ileum, and on the left side from the splenic flexure to the pelvic colon. Partial colectomy was generally preferred for these injuries.

The ileo-colic spur, or "gun-barrelling" of transverse and pelvic colon, was made at least four inches long. The clamps were left on the exteriorized ends for twenty-four hours. The gut was brought out through the medial end of the transverse incision. It was not sutured to the abdominal wall.

Reference has already been made to the placing of tubes for sulphadiazine to the upper end of the lesion—that is, to splenic or hepatic flexure. If there were gross peritoneal infections or an associated intraperitoneal hæmorrhage which was thought to be infected, the pelvis was drained. (Illustrative cases: LXXV, XCV, CXX and CLXVII.)

Wounds of the Mesocolon.

Wounds of the mesocolon were present as the only lesion in four cases. One death (Case CLVII), the result of an infarction in the transverse colon and of many other coincident intraabdominal injuries, has already been mentioned.

In two cases wounds of the mesocolon required suture. But in neither of these was the blood supply of the gut affected.

A fourth case is included in this category, although no definite wound of the mesocolon was found.

In this a wound of the left loin caused symptoms suggestive of intraperitoneal injuries. At operation a large retroperitoneal hæmatoma extending into the transverse mesocolon was found in the mid-upper abdomen. The middle third of this segment of gut was dusky, and on account of this a transverse colostomy was done.

Contusions of the Colon.

The danger of secondary rupture in contusions of the colon in which this organ is not put at rest by a colostomy either proximal to the contusion or actually in the affected segment, is well illustrated by Case XXX.

At operation this man was found to have three small ecchymoses of the pelvic colon and some blood in the peritoneum. Palpation of the affected loop revealed no foreign body, nor could any tear in the peritoneal coat of the bowel be detected. The abdomen was closed without drainage. The team moved forward to another main dressing station within a day or so, and this man was left behind. On a visit to see him about the seventh post-operative day, he was found to be sick and his abdomen to be distended. His temperature had risen suddenly on the fifth day and he had complained of sudden severe abdominal pain, which had subsequently become easier. A pelvic peritonitis, from an infected hæmoperitoneum, was diagnosed. He died forty-eight hours later. At autopsy a small foreign body was found, lying close to a perforation of the pelvic colon, in the area of an ecchymosis.

By contrast, Case CXXVI demonstrates the efficacy of this precaution.

Here a second foreign body lodged in the rectal wall, the main damage being caused by a piece of shell passing through the pelvic colon and destroying the mid-portion of the small intestine. This second foreign body was found only at post-mortem

examination some five weeks later. Left inguinal colostomy had been performed and the fragment in the rectal wall, submucosal, was walled off, with no abscess round it. Death was due to infective endocarditis.

Rectum.

There were six cases of injury to the rectum, with one recovery. Intraperitoneal injury was present in two cases. Extraperitoneal injury was present in three cases and in one of these recovery occurred. Combined extraperitoneal and intraperitoneal injury was present in one case.

In addition to this anatomical classification, rectal wounds separated themselves into two clinical groups which have a very different mortality: Group A, those with small wounds and little muscle damage; Group B, those with large shattering sacral wounds and associated muscle damage in the buttocks.

Group A.—There were three cases in Group A. Two consisted of gunshot wounds (perforating) of the buttocks and one of a shell wound of the right iliac fossa. Although only one of these patients recovered, it is felt that this class of wound should have a mortality somewhat comparable to colon wounds. These three cases all occurred very early in our tour of duty, and before any suction or sulphadiazine was available.

Two patients had intraperitoneal injuries and arrived late after wounding (twelve to twenty-four hours); and one had an associated injury of the small bowel which required resection. Both of the patients died in ileus after forty-eight hours. The third (Case VIII), the only patient who recovered, had a through-and-through extraperitoneal gunshot wound in the middle third of the rectum. In his case left inguinal colostomy was carried out, and a large pelvic extraperitoneal hæmatoma was drained. Wounds were not excised.

Group B.—There were three cases in Group B. In all of these there were extensive buttock wounds with destruction of the lower half of the sacrum and coccyx. All the patients had large defects of the posterior part of the rectum and in one instance there was an intraperitoneal lesion. The casualties all occurred in the Alamein battle, and so the patients were treated in the Phase 2 series of cases. Sacral and buttock wounds were widely excised and the resultant cavities were dusted with sulphonamide powder and dressed with "Vaseline" gauze. In each case a left inguinal colostomy with a spur was immediately instituted. In addition the intraperitoneal perforations were closed with a double layer of sutures and sulphadiazine was injected through a tube down to the pelvis.

It was decided that these patients should be the only "abdominal" casualties that should be evacuated as soon as they were fit to travel, for it was inevitable that the wounds would become infected. It was felt that the patients would travel better before they began to suffer from a general toxæmia. Moreover, there were few facilities for adequate dressings of large infected wounds and patients with large buttock wounds could not be made very comfortable in the field.

The man with the combined lesion died suddenly in forty-eight hours following a rigor; probably death was the result of a septic embolus. The other two were evacuated quite satisfactorily on the third day directly to a base hospital, some seventy miles away. They both died in the third week of toxæmia from the extensive buttock wound. Case CLIV is quoted only because the patient was seen at an Australian general hospital just prior to his death.

Mortality in this class of rectal case must inevitably be high. It is felt, however, that early evacuation was justified to a centre where skilled nursing and all facilities for dressing and comfort are available.

SUMMARY.

1. The experience of 90 abdominal wounds, operated on in forward areas, is used as a basis. Certain cases are quoted.

2. General management in all stages is reviewed, and standards are laid down under which the mortality was halved.

3. Benefits of rapid transfusion are shown.

4. Intraperitoneal injection of sulphadiazine would seem, clinically, to reduce peritoneal infection following bowel perforation.

5. The necessity is shown for routine early post-operative gastric suction and continuous intravenous therapy in all cases of perforated bowel.

6. Lesions of various organs are reviewed in detail and their management is discussed.

7. Wounds of the colon, if "exteriorized", are no more fatal than those involving small bowel. A recovery rate of at least 70% is to be expected if patients come to operation within twelve hours.

ACKNOWLEDGEMENTS.

The illustrations have been kindly drawn for me by Miss Joan Devine.

REFERENCES.

(1) C. Naunton Morgan: "A Discussion on Factors which Make for Safety in the Pre-Operative and Post-Operative Treatment of Carcinoma of the Rectum: Post-Operative Treatment", *Proceedings of the Royal Society of Medicine*, Volume xxxiv, Part 2, September, 1940.

(2) J. Smith: "Surgical Experiences in the Middle East", THE AUSTRALIAN AND NEW ZEALAND JOURNAL OF SURGERY, Volume xi, 1941-1942, page 153.

(3) H. B. Devine: "Colon Surgery in the Debilitated", *The Journal of the College of Surgeons of Australasia*, Volume i, November, 1928, Number 2, page 173.

(4) H. B. Devine: "Safer Colon Surgery", *The Lancet*, March 21, 1931, page 627.

(5) W. H. Ogilvie: "The Treatment of Abdominal Wounds", *Journal of the Royal Army Medical Corps*, December, 1941, Volume lxxvii, page 299.

APPENDIX.

Brief Notes on Cases to which Reference is Made in Text.[1]

Phase I.

Case III.—G.S.W.; perf.; en. R.U.Q.; ex. R. loin; hæmaturia; resus. nil; opn. 6 hrs.; 2 small perfs. sp.F. closed; large retrop.H. to caecum; drain M.P. and H.; I.V.F. 24 hrs.; sulph. 15 gm. by mouth; recovery.

Case VII.—0900 hrs.; B.W.; pen. R. loin; M.D.S. 1400 hrs.; very shocked, 2,000 c.cm. B. at 1900 hrs.; pulseless at 2100 hrs. and further 1,000 c.cm. of B.; pulse palpable; mictn. = pure blood; large mass in R. loin; 2200 hrs. R. nephrectomy; finding = kidney transected and tear through pedicle; 2225 hrs.; death.

[1] Abbreviations used (alphabetically arranged) are as follows: A.C. = ascending colon. B. = blood (amounts are those given as pre-op. resus. at M.D.S.). B.W. = bomb wound. C. = colon. D.C. = descending colon. En. = entrance. Ex. = exit. Exc. = excision. F. = flexure. F.B. = foreign body. G.B. = gall-bladder. G.S.W. = gunshot wound. G.S. = glucose saline. H. = hæmatoma. Hæm. = hæmorrhage. H.F. = hepatic flexure. I.V.F. = intravenous fluids. L. = left. L.I.F. = left iliac fossa. L. umbi. = left umbilical. M.P. = Morrison's pouch. Opn. = operation. Pen. = penetrating (entrance wound only). Perf. = perforating (entrance and exit wounds). P.C. = pelvic colon. P.O. = post-operative. Pre-op. = pre-operative. R. = right. Resus. = resuscitation. Retrop.H. = retroperitoneal hæmatoma. R.U.Q. = right upper quadrant. Rpt. = repeated. Ry. = recovery. S. = serum. S.F. = sigmoid flexure. S.I. = small intestine. Sp.F. = splenic flexure. Suction = continuous gastric suction. S.D. = sulphadiazine. Sulph. = sulphonamide. Sulph.Vas. = sulphonamide and "Vaseline". T.C. = transverse colon. W. = wound. Time expressed after opn. is approx. time between wound and operation.

Case VIII.—G.S.W. perf. post. to R. and L. trochanters; blood P.R.; retention; L. sciatic paresis; lower abd. rigid; severe shock; 1,300 c.cm. B. given; opn. 10 hrs.; small amount blood peritoneum; C. full of blood; very large retrop.H. filling pelvis; L.I. colostomy and drainage H. on each side; sulph. 15 gm. per orum; colostomy opened and melena 4 days; recovery.

Case XXX.—S.W. pen. L. loin; no resus.; opn. 10 hrs.; some free blood, holes in P.mesoC. and contusion of P.C., and retrop.H. of true and false pelvis; drainage extrap.; P.O. abd. pain; T. 105° on 5th day; draining freely on 8th day, but still pain; enema = poor result; death on 10th day; P.M. perf. P.C. and F.B. lying free close by.

Case XXXI.—B.W. pen. L. flank; v. exsang.; dull L. flank; 2,500 c.cm. B. given; opn. 10 hrs.; severed splenic pedicle; splenectomy; P.O. 1,500 c.cm. B. given; death; P.M. no further hæm. or overlooked injury.

Case XXXVIII.—S.W. pen. R. loin; fæcal discharge; v. shocked; 1,600 c.cm. of B. and 550 c.cm. of S. given; opn. 22 hrs.; 2 large intrap. tears of asc. colon, large extrap. P. of flexure, retrop.H.; R. colectomy; P.O. I.V.F. for 3 days; ileostomy acting after 24 hrs.; recovery.

Case XLVI.—1700 hrs.; B.W. pen. L. umbi.; 1900 hrs. M.D.S.; condition: Sl. rigidity L. rectus; P. 78; 2100 hrs. = nausea, P. 96; 2130 hrs. P. 104 and opn.; no fluid or gas in peritoneum; omentum and T.C. presenting; 3 small perfs. of T.C. closed and F.B. felt and removed from colon; sulph. powder; abd. closed and drain to rectus; P.O. I.V.F. 24 hrs. 2,000 c.cm., 0·25% sulph. in G.S.; recovery.

Case LIII.—B.W. pen. L. lower chest; 1,000 c.cm. of S.; opn. 3 hrs.; S.F. mobilized; 5 perfs. closed, mesocolon sutured, trans. colostomy, drain pelvis, tube to flexure and 3 injections of S.D., suction and I.V.F.; death 5th day; P.M. injured and devitalized colon, collapsed L. lung.

Phase 2.

Case LXXV.—R. subcostal S.W. pen.; hæmaturia ++; 1,500 of S. and 1,000 c.cm. of B.; opn. 10 hrs.; B.P. 130/80; hepatic F. damaged, destruction lower pole R. kidney; R. colectomy, R. nephrectomy; tube and 3 injections of S.D. to M.P.; P.O. ileostomy functioned in 24 hrs.; I.V.F.; suction 3 days; recovery.

Case XCV.—L. lumbar S.W. pen.; compd. fract. mandible; 1715 hrs. M.D.S.; 2000 hrs. no abd. tenderness or rigidity; 2130 hrs. vomited, R.U.Q. rigid, hæmaturia, P. rising, resus. S. 500 c.cm. and B. 1,000 c.cm.; 2200 hrs. opn.; retrop.H.; a little blood in peritoneum; desc. C. mobilized; large extrap. perfs.; laceration lower L. kidney; L. colectomy and nephrectomy; drain to pelvis; 3 injections of S.D. to kidney bed; P.O. clamps off 24 hrs.; colostomy acted 6th day; I.V.F.; suction 7 days (chloride controlled); no œdema, but polyuria 90 oz. av. last 3 days; recovery.

Case XCIX.—Mult. S.W.'s abd.; prolapse ½ small gut and many transections; mult. S.W.'s thighs; hæm. ++ from abd.; moribund; resus. 2,500 c.cm. of S. and 4,500 c.cm. of B.; opn. 6 hrs.; B.P. 100/50; resection at least ½ S.I.; large F.B. in mesentery removed; ant. abd. wall repaired; S.D. to upper abd. and rpt.; drain to pelvis; exc. thigh wounds; ligation L. deep fem. A.; during opn. 1,500 c.cm. of B. and 1,500 c.cm. of S.; P.O. B.P. 90/50; suction I.V.F. inc. S. 1,000 c.cm. daily; death 56 hrs.; P.M. L. ant. and post. subphrenic abscess, perf. stomach (high in cardia) from small undetected wound L. flank, perf. chest and diaphragm, 3 pints blood L. chest.

Case CXII.—G.S.W. pen. L.I.F.; mod. shock; 1,000 c.cm. of S. and 1,000 c.cm. of B.; opn. 4 hrs.; ligation R. inf. epigas. art.; repair transected ileum and closure other perfs.; blind end caecum destroyed; caecum fixed by post. appendiceal adhesions; lac. closed and sutured extrap. with drain to it; pelvic tube and S.D. and rpt.; P.O. I.V.F. and suction 4 days; fæcal fistula at gen. hosp.; spontan. closure in 6 weeks; P.O. secondary hæm. from R. inf. epigas. art.; recovery.

Case CXVII.—Pen. S.W. R. chest.; mod. shock; 1,000 c.cm. of S and 1,000 c.cm. of B.; board-like rigidity; max. tender R.U.Q. and L.I.F.; opn. 8 hrs.; perf. liver and G.B.—cholecystostomy; perf. trans. C.—colostomy; 3 perfs. jejunum, perfs. closed; F.B. in gut removed; S.D. to M.P. and rpt.; P.O. I.V.F., suction 5 days; recovery.

Case CXX.—S.W. pen. R. lower chest; 2,000 c.cm. of S. and 1,000 c.cm. of B.; opn. 9 hrs.; large holes in H.F. intrap. and retrop.; R. colectomy and ileo-colostomy; large perf. extrap. duodenum on R. and post. of 2nd part closed; death on table of resp. failure.

Case CXXIV.—S.W. pen. R. chest; mod. shock; 1,800 c.cm. of B. and 500 c.cm. of S.; opn. 15 hrs.; large W. R. upper liver—packed; hole diaphragm admits finger—closed; drain M.P.; chest W. (sucking)—closed over sulph. vas. pack; I.V.F. 24 hrs.; recovery.

Case CXXV.—Mult. B.W.'s, one R. lower chest; mod. shock; 1,000 c.cm. of B.; pain unrelieved by morphine; abd. board-like; opn. 9 hrs.; choleperitoneum from perf. G.B.; stomach v. dilated; cholecystostomy; drain M.P. P.O. I.V.F. and suction 48 hrs.; recovery.

Case CXXVI.—S.W. pen. perineum, H. of penis; 1,000 c.cm. of B.; opn. 7 hrs.; mid-half S.I. destroyed; resection, 4in. spur and ileostomy; colostomy for perf. pelvic C.; large extrap.H. round bladder—drained; drain pelvis; S.D. and rpt. twice; P.O. I.V.F. and suction 3 days; ileostomy acted in 24 hrs. and colostomy in 48 hrs.; prolapse of ileostomy after 4 days, but was reduced; evac. 7 days; spur crushed about 3rd week without effect; I.V.F.'s given; becoming emaciated; bowel short-circuited, but still losing contents; death in 5th week; P.M. infective endocarditis, secondary to venous thrombosis of arm; F.B. found in rectal wall posteriorly; no perf. or inflam. round F.B.

Case CXXVII.—S.W. perf., en. suprapubic, ex. L. sacro-iliac; severe shock; 1,000 c.cm. of S. and 1,500 c.cm. of B.; opn. 8 hrs.; mult. perfs. jejunum and ileum—closed; 2 small holes P.M.C. Peritoneal cavity suddenly filled with blood. Tangential W. L. int. iliac vein found; vein ligated; intrap. hole bladder closed; extrap. hole enlarged for cystostomy; drain pelvis and S. of Retzius; 1,000 c.cm. of B. during opn.; S.D. and rpt.; P.O. I.V.F.; suction 3 days; recovery.

Case CXXXIII.—G.S.W. perf. R.-L. mid-axilla subcostal; severe shock; 2,000 c.cm. of S. and 2,000 c.cm. of B.; opn. 9 hrs.; intrap.H. +++; 2 high jej. perfs.—closed; large mesenteric H. with perf.; perf. trans. mesoc. at root; bubbles air from duodenum—mobilized and 2 extrap. perfs. closed; R. perinephric H.—not drained; drain pelvis; S.D. to jej. rpt. twice. P.O. I.V.F. and suction 5 days; recovery.

Case CXXXIV.—G.S.W. perf. L. buttock, R. thigh; hæmaturia; 2,000 c.cm. of S. and 1,500 c.cm. of B.; opn. 8 hrs.; blood in peritoneum and peas and liquid fæces; peritoneum injected; H. filling both lat. pubic walls; large tear lower ileum—closed; drain pelvis; S.D. and rpt.; bladder opened and full of clot, and steady hæm. from 2 holes at base; cystostomy; drain to each paravesical space; P.O. I.V.F. and suction 5 days; P.O. pneumonia; recovery.

Case CXXXV.—S.W. pen. L. epigastric; slight tenderness; no rigidity; no shock; opn. 7 hrs.; track followed = perf. peritoneum; laparotomy; perf. liver; prepyloric perfs. stomach—closed; P.O. I.V.F.; suction 5 days; P.O. pneumonia; I.V. sulphathiazole till off suction, then "M & B 693"; recovery.

Case CXXXVIII.—S.W. pen. L. chest 8 inches long about 6th rib, ant. lat.—sutured at R.A.P.; appears to "blow" occas.; abd. sounds lower base; severe shock; 3,000 c.cm. of B.; opn. 10 hrs.; sutures out, débris and fragments ribs removed; ruptured stomach presented; large lac. sutured; L. lung contused; laparotomy; reduce hernia; close diaphragmatic defect; further lac. near fundus found and closed, after F.B. removed from stomach; large F.B. free in peritoneal cavity; spleen—bruised; ecchymoses in T.M.C.; P.O. I.V.F.; suction 5 days; 48 hrs. P.O. pneumonia; I.V. sulphathiazole; evac. B.G.H. 7th day—irrational—cyanosed—commencing fluid L. base, ? empyema; death; P.M. "pus round upper L. diaphragm; consolidation L. lung; carnification R. lung; scme sanguin. fluid in peritoneum; all sutures solid; jejunum = black from interstit. hæms. (not regarded as of significance)".

Case CXLIII.—S.W. pen. L. flank; hæmaturia; mod. shock; 1,000 c.cm. of S. and 1,500 c.cm. of B.; opn. under 12 hrs.; peritoneum full of blood; contusions of S.F. and D.C.; bleeding from mesocolon; L. perinephric H.; colon mobilized, no perfs. found; colostomy of affected loop; lower pole L. kidney destroyed; L. nephrectomy; drainage; colostomy opened in 48 hrs.; P.O. no I.V.F.'s; recovery.

Case CLI.—G.S.W. perf.; en. L. umbi., ex. L.5; 800 c.cm. of S. and 1,500 c.cm. of B. Opn. 6 hrs.; perf. desc. colon; colostomy after mobilizing; perfs. jejunum closed; S.D. to splenic region—rpt. once; P.O. I.V.F.'s and suction 5 days; recovery.

Case CLIV.—S.W. shearing L. buttock; destruction coccyx and lower half sacrum; fæcal discharge; 400 c.cm. of S. and 1,000 c.cm. of B.; opn. 6 hrs.; L.I. colostomy—no intrap. lesion; wide excision mm.; removal of large F.B. and pieces of sacrum; rectal ampulla exposed and has large tear; tracks to L. obturator ext. and under R. glut. max. widely opened; drain tubes to these; sulph. powder; "Vaseline" gauze over wounds and dressing; P.O. I.V.F.'s 48 hrs.; evac. hosp.; on 14th day seen at hospital; V. toxic; wound fairly clean; colostomy acting; R. leg a bag of subcut. pus; death.

Case CLVII.—G.S.W. perf., en. L. epigas., ex. lower dorsal, almost mid-line. Gas and blood from ant. wound; hæmaturia; 1,500 c.cm. of B.; opn. 10 hrs.; L. lobe liver in fragments; 2 large perfs. in A. and P. stomach; severed mid-colic art.; infarction trans. colon; large perinephric H.; closed as hopeless; death in 4 hrs. *N.B.*—This was only case brought to operation in which no attempt at repair was made.

Case CLIX.—G.S.W. pen. L. lower chest; 1,000 c.cm. of B.; opn. 10 hrs.; two perfs. in 1st 2 inches jejunum—closed; two perfs. in post. wall stomach—closed; large retrop.H. round aorta; opn. notes state "bullet cannot be felt, but probably lies near aorta"; P.O. I.V.F.'s continued; reported by orderly 6 hrs. P.O. "patient in good condition when he suddenly coughed and died"; probable aortic rupture; no P.M. practicable.

Case CLXVII.—G.S.W. perf. R. flank-L. flank; paresis L. leg; fæcal disch. R. wound; severe shock; 1,500 c.cm. of S. and 2,000 c.cm. of B.; opn. 12 hrs; large tears intrap. and extrap. A.C.; R. colectomy with ileo-colostomy; S. intestine intact; large H. felt beneath D.C. and intrap. hole of colon; L. trans. incision; D.C. mobilized and colostomy with spur of affected segment; drain H.; S.D. to M.P. and rpt. twice; P.O. suction 5 days, I.V.F.'s 5 days; evac. base; death 12th day; cellulitis R. ant. abd. wall.

Surgery in Other Countries.

[In this column will be published short résumés of articles likely to be of practical value from Journals published in other countries and not readily accessible to surgeons in Australia and New Zealand.]

A SIMPLE METHOD OF INTRAARTICULAR ARTHRODESIS OF THE HIP JOINT.

Günther Imhäuser (Leipzig): *"Über eine intra-artikuläre Spanarthrodese des Hüftgelenks"*, *Zeitschrift für Orthopädie*, 1941, page 48.

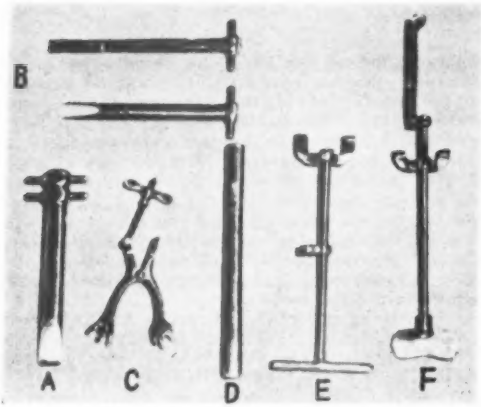

FIGURE I.

CONSERVATIVE methods of treatment of severe arthrosis of the hip joint too often fail to prevent the development of painful persistent adduction deformity. In patients of not too great age, in whom the condition is unilateral and consequent on some juvenile defect, an arthrodesis with the joint in good position will eliminate these disabilities; and Imhäuser in this paper gives details of a simple and effective method of performing this arthrodesis, which has been worked out by Professor Schede, at Leipzig.

The principal instrument required is the "tubed chisel" of Novotny, which, with other accessory instruments devised by Schede, is shown in Figure I. The contracture must first be overcome, if necessary by tenotomy of the adductor tendons and by mechanical or manual correction. The hip region is then covered with sterile towels and the regional anatomy studied by palpation, with the help of a previously taken X-ray film. By this means the proper point of entry of the Novotny chisel and the direction it should follow are established. A small longitudinal incision, made some centimetres above the trochanter, and about two centimetres long, allows the chisel to be passed through the skin and down to the bone. Position and direction are now checked by an X-ray film, and the chisel is then driven in some six or seven centimetres. Meanwhile an assistant has been taking from the surface of the tibia a graft about six by two centimetres, and this is pushed down the tube of the Novotny instrument after the blade of the chisel has been removed, and is driven home with a few light taps of a wooden mallet. The tube of the chisel is then withdrawn by means of a special screw apparatus shown in Figure I, E or F. A

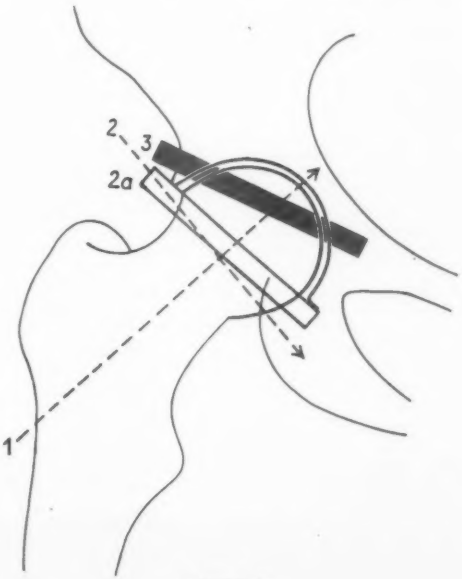

FIGURE II.

further X-ray film checks the depth and position of the graft. The hip in a successful case should be completely immobile. A double plaster spica is applied to the pelvis and thighs.

Eight weeks later a plaster in which the patient can stand up is put on, and this remains in place for two to four months, according to the X-ray indications of ankylosis. The direction in which the graft is laid is important. It should pass through both margins of the acetabulum as well as the head of the femur. Figure II shows diagrammatically the various factors. Of these, the line 2 is not effective, some adduction deformity being still possible; 2*a* is good, but 3 is ideal. In the first of the six cases reported the results fell short of the desired goal, owing, Imhäuser states, to too short a period of primary immobilization. In later cases this fault has been corrected, and the results show a corresponding improvement.

ARTHUR E. BROWN.

A DIATHERMY KNIFE FOR RECTAL STENOSIS.

R. von Oppolzer (Vienna): *"Ein Rektalstenosen-Diathermiemesser"*, *Zentralblatt für Chirurgie*, Volume lxviii, April 26, 1941, page 784.

IN rectal stenosis due to scar tissue following operations involving circular suture of the anal canal, treatment by dilatation with bougies is very tedious and often painful, and the result is only too often a pencil-narrow anal ring, capable almost of producing obstruction. Reoperation on these strictures is difficult and carries the danger of secondary fæcal fistula, or of recurrence of the stenosis. Oppolzer has devised a diathermy knife by means of which the ring of scar tissue which extends at most upwards for a distance of one centimetre, can either be removed or widened by a number of radial incisions.

The knife is introduced through a short rectoscope under direct vision, and its blade is carried above the stricture, from which position the necessary incisions are made in a downward direction; thus danger to the bladder wall is avoided. Smoke produced is sucked out by means of the rectoscope attachment. Oppolzer speaks highly of the instrument and of its results in his hands.

ARTHUR E. BROWN.

TREATMENT OF ACUTE OSTEOMYELITIS BY ABSCESS INCISION WITH CHEMOTHERAPY AND SEROTHERAPY.

Curt Hempel (Fritzlar): *"Zur Behandlung schwerer akuter hämorrhagischer Osteomyelitis mit Abszessinzision und Sero- und Chemotherapie"*, *Zentralblatt für Chirurgie*, Volume lxviii, April 19, 1941, page 741.

LITTLE unanimity has yet been achieved in the question of the best treatment of acute osteomyelitis in its severer forms. Of patients with the fulminating type in Eiselsberg's clinic all died within seventy-two hours; for this type surgery of any sort seems to have little to offer. But in those cases of less primary severity the treatment advocated seems to vary from the radical procedure of complete resection of the shaft of the bone to a purely conservative attitude.

Simple incision of the abscess has given a mortality of 10% among 220 patients at Heidelberg, and one of 7% among 176 patients in the clinic of Wilms. When bone chiselling has been part of the primary operation, Enderlen reported a mortality of 14% among 70 patients, and it is recorded that metastatic abscesses and grave complications of other sorts are twice as frequent when the chisel is used. In general, the high expectations based on the more radical procedures have not been realized. Bier claims that he has obtained better results with passive hyperæmia than with any other method. He combines with it repeated puncture of the abscess, but no drainage of it. Moog, from Kiel, reports results disappointingly the reverse of those of Bier.

In general it would appear that in the primary "septic" cases the patients are doomed. In others the question as to why any particular line of treatment should be successful in some cases and fail in others, is still to be answered. The grade of infection is important, but we have no means of estimating it, and it is revealed only by the future course of the disease. Those patients who do not succumb within a day or two are those suffering from a less severe type of infection, and in these cases there is a definite possibility of assisting the body defences by therapeutic measures. The actual method of dealing with the abscess in these circumstances is probably of minor importance, but it seems that incision, if carried out too early, raises the general mortality.

Animal experiments with staphylococci would seem to show a reaction very much on a parallel with the course of acute osteomyelitis in man—either swift sudden death or a reasonable degree of recovery—and the difference seems to lie in the powers of body resistance. The fatal effect of the staphylococcal infection is based on the action of a

complex but specific toxin, which can be neutralized by a specific antitoxin. In animal experiments immunization is swiftly followed by a massing of polymorphonuclear and mononuclear leucocytes which, owing to neutralization of the bacterial toxins, can develop their phagocytic powers. This effect must not be lost sight of when better results in treatment of human patients are desired. Serum treatment must be used at the earliest possible moment. The earlier the protective serum can be introduced, the more chance there is of limiting the damage done. Early bacteriological diagnosis is therefore important, and since a later mixed infection holds special danger for the patient, the bacteriological study must be often repeated, especially when new abscesses form or joint suppuration occurs. The antitoxin appropriate to the results obtained must then be exhibited. This point is important if failure of the method is not to be wrongly estimated.

Not only is early giving of the serum necessary, but it must be given in frequently repeated doses. The concept must always be kept in mind that passive immunization is short-lasting, and cannot be expected to repair any damage already done. The object of each dose of serum, then, is to neutralize freshly formed toxin before it forms its harmful and irreversible union with the body cells, and the need for repeated doses becomes evident.

The difficulty of making a true assessment of the value of serum treatment is evident, so many important factors being unknown. It is true that the treatment is not always successful, but whether the failures are due to variations in the strains of infecting staphylococci is not known, and must be the subject of future research. The really fulminating cases are rare, and only a few of them are reported in detail. Therefore estimate of the value of the method must at present depend on the clinical results observed. When so experienced an observer as H. Schmidt states that not many types of serum have the power of saving life in occasional desperate cases that antistaphylococcal serum has, the clinician is well advised to give it clinical trial.

Parker and Shands, in cases of acute osteomyelitis, gave a first dose of 20,000 units intramuscularly to children under ten years of age, and 40,000 to older children. If no unfavourable reaction supervened, they gave a further 40,000 to 60,000 units by the continuous intravenous drip method. Some workers use the serum of convalescents. Schmidt treated the relatives of a patient with vaccine, and from their blood produced a serum which helped him to save a patient almost hopelessly ill. Hempel himself, after losing two patients of severe grade infection, in spite of incision of the abscess and the exhibition of "Uleron", treated his next similar patient with serum in association with these measures. This child, whose history is set out, survived.

ARTHUR E. BROWN.

THE TREATMENT OF NEURITIS FOLLOWING GUNSHOT WOUNDS.

Fr. Heck (Würzburg): *"Zur Behandlung der Schussneuritis"*, *Zentralblatt für Chirurgie*, Volume lxviii, Number 22, 1941, page 1031.

THE Reserve Military Hospital II at Würzburg specifically admits patients suffering from nerve injury, and therefore has had to handle large numbers of them. It has been found that the problem of intractable pain following nerve injury is a much more difficult one to solve than is the actual restoration of the nerve after damage. The intense pain suffered by such patients has been a stimulus to a search for its remedy. The patients all showed increased sweating, shiny skin, and nail changes. The causalgia was so severe that there was a constant demand for ice for them; and one man with combined median, ulnar and radial nerve injury could do nothing but sit all day by the basins, letting the cold water run over his arm. Mental stress showed itself in grave forms.

Causalgia is a pain that does not remain localized, but characteristically radiates, which is evidence that it is of sympathetic rather than peripheral nerve origin. Therefore surgeons must consider the sympathetic tracts in their fight against the pain. If simpler methods fail to give relief within a reasonable time, the surgeon must be prepared to attack the sympathetic source of origin by surgical means. The method of attack can be either directed against the outer coats of the main arteries to the affected limbs or against the paravertebral chain, the second, third and fourth lumbar ganglia in the lower limb and the stellate ganglion in the upper limb. Although several authors have expressed disappointment with the results of arterial stripping, there are a number of very favourable reports. Leriche and Fontaine, in a series of 1,256 sympathectomies, reported consistently good results from periarterial sympathectomy, and some of their patients were followed for up to eight years. With the knowledge that the vessel-stripping operation is a much simpler one than a ganglionectomy, it is right to try it first. This was done in the three cases which Heck reports in this paper.

In the case of the first patient, a soldier with severe muscular destruction in the right thigh, the pain set in simultaneously with wound healing. No evidence of actual

damage to the main nerves was elicited, but all means of alleviating his pain were unsuccessful. The femoral artery was stripped of its adventitia for a distance of 15 centimetres, and a strip 12 centimetres long was excised. Immediately the pain ceased. Appetite and general condition improved. A month later the patient was discharged from hospital and reported himself later as doing well. In the second case, stripping and denudation of the axillary artery were equally successful. In both these cases, after a few weeks of complete ease, some minor return of symptoms occurred, the pain being intermittent and easily controlled by medical means. In both cases a further attack on the sympathetic chains was planned should it be indicated; but in neither of them has it had to be done.

In the third case the relief of pain lasted only three days and then gradually the causalgia returned in all its old force. The right lumbar sympathetic chain was exposed and seen to be affected grossly by inflammatory changes. The second, third and fourth lumbar ganglia were removed, since when only occasional transitory pains have occurred.

Causalgia is held in this paper to be a secondary phenomenon and associated in some way with wound healing. The correct point of attack is the sympathetic lines of supply; of the operations, that on the adventitia of the large vessels is the simpler and may in itself be sufficient to produce cure.

ARTHUR E. BROWN.

SURGICAL TREATMENT OF PEPTIC ULCERS.

F. Hohmeier (Coblenz): "*Zur Magen Chirurgie*", *Zentralblatt für Chirurgie*, Volume lxviii, Number 22, 1941, page 996.

HOHMEIER, in discussing the surgical treatment of perforated peptic ulcer, states that he has abandoned the use of resection of the stomach on both technical and general grounds. He now relies primarily on simple oversuture of the ulcer where possible. Should this simple procedure not appear to be sufficient, he dislikes the principle of reinforcing it by omentum, because of the difficulty this causes when future secondary operations are necessary. He considers that the likelihood of a secondary operation is so great that all patients should be warned that they will later have to subject themselves to one. If more than simple suture is needed, an immediate palliative gastro-enterostomy is the method of choice; but though the posterior route has ordinarily very evident advantages, it is not preferred in these cases because of the difficulties it puts in the way of successful secondary operation. Hohmeier's principle is to make an anterior antecolic anastomosis; but since this does not eliminate the danger of vicious-circle vomiting, even when the afferent and efferent loops are anastomosed, he accompanies the gastro-enteric anastomosis with an anastomosis "en-Y" after Roux. He states that he has never seen an anastomotic ulcer develop with this technique, and that the result in regard to the patient's comfort is excellent. The technique of performing the operation is easy, and it leaves the problem of later resection, should such prove necessary, a very simple one. He is emphatic that this is the procedure that should be adopted in cases of perforated ulcer should any primary treatment beyond that of simple suture be necessary.

ARTHUR E. BROWN.

THIEMANN'S DISEASE.

O. Dyes (Munich): "*Spätform der Thiemann'schen Krankheit*", *Zentralblatt für Chirurgie*, Volume lxviii, Number 22, 1941, page 1036.

THE case is reported of a twenty-three year old man in whom the middle joints of both third fingers had in the past three years become crooked, swollen and immovable, along with intermittent radiating pain of a burning type. The pain seemed to arise spontaneously, and not to be caused by active use of the hand. An X-ray film showed that the deformity arose in the bone and joint surfaces, and was not periarticular. The heads of the proximal phalanges and the bases of the second phalanges in the affected finger on each hand were enlarged and broadened, without irregularity. The change was noticeable in the bone cortex only, the medullary cavity being unaffected. A very pronounced enlargement of a cup-shaped type was evident in the joint surface of the base of the second phalanx. The joint surfaces were somewhat uneven, and the primary seat of the disease was apparently here, the broadening and other changes in the bones being apparently secondary to joint changes.

There did not appear to be any inflammatory condition present, and the lesion could easily be grouped among the loose group of *arthrosis deformans* without further effort to elucidate the pathology. Thiemann, however, in 1909 described a disease in the basal

epiphysis of the second phalanx of the middle finger, which Dessecker later rightly grouped among the "epiphyseal necroses". Thiemann's description covered all the factors appearing in this case, including its bilateral appearance in a young man. The epiphyseal plates are broader than usual and concave in the middle, almost split into two parts, and thus they have the appearance of a meniscus. The disease generally commences after the twelfth year, and reaches its peak between the fifteenth and eighteenth, with healing occurring at the time of cessation of bodily growth. It leaves behind it a massive thickening of the shaft near the epiphysis and a shortening of the basal phalanx.

There are practically no reports in the literature of cases of severe finger deformity which are recognizable as a late result of this disease, although Thiemann himself forecast that such might be expected. This case is one such. More often the epiphyseal necrosis heals so well that a nearly normal joint is left, as is now known from the work of Köhler and Perthes.

The onset of symptoms in the patient reported is later than usual, being in fact at about the time when Thiemann's disease should be healing or healed—namely, twenty years. But the onset of symptoms also corresponded with the lad's commencing his period in the labour service of Germany, with its heavy manual labour. The history is unusual in this aspect, but it is possible that previous pain was neglected. The form of the bone ends suggests that the process of new bone formation had ceased, and the shortness of the middle phalanx and the limitation of the deformity to the ends of the bones indicate that it was a disease of adolescence.

ARTHUR E. BROWN.

A TECHNIQUE FOR EXPOSURE OF THE LUMBO-SACRAL PLEXUS IN GUNSHOT INJURY OF THE PELVIS.

W. Tönnis (Berlin): *"Operative Freilegung des Plexus lumbo-sacralis bei Schussverletzungen des Beckens"*, *Zentralblatt für Chirurgie*, Volume lxviii, Number 22, 1941, page 1007.

THE work of König and of Iselin and Guleke on the exposure of the sciatic nerve in the buttock does not carry this exposure above the infrapyriform foramen; and Tönnis sets forth in this paper a procedure which gives good exposure deep to this point.

In a described case the original operation disclosed the peripheral end of the severed sciatic nerve lying lateral to the biceps and engaged in a thick mass of scar tissue. The proximal end was not found at all. The operation was therefore suspended, while a further line of attack was evolved whereby a successful suture of the nerve might be attained. Twelve days later a second operation was performed, under spinal anæsthesia. The patient was placed lying on his face with his legs separated. The incision started at the posterior superior iliac spine, and curved round the medial and lower margins of the *gluteus maximus* muscle. The coccyx and the terminal segment of the sacrum were resected, and the *gluteus maximus* was separated from the sacrum along its lateral border. The sacro-tuberous ligament was thus exposed and severed, after which the pyriformis muscle was easily defined, and was cut through transversely about a finger's breadth from its origin. This step laid clearly open to view the lumbo-sacral plexus and the origin of the sciatic nerve from it. On retraction of the gluteus laterally the proximal end of the injured nerve was dissected free from a mass of scar tissue in the pyriformis foramen. The site of injury was three fingers' breadth above this foramen, and a good thumb width below the origin of the nerve from the plexus. Strong hyperextension of the hip, with extreme flexion of the knee, enabled the two ends of the nerve to be approximated and sutured.

The operation was performed on October 19. On November 23 the retaining plaster splint was removed. On February 10 next the patient claimed that he could feel pins and needles in the sole of his foot; and a month later return of function in the posterior muscles of the thigh was becoming evident.

In two other cases of evident sciatic nerve severance the above-described operation was carried out in one stage as a primary procedure. When the plexus roots had been displayed, the incision was carried round the lower border of the *gluteus maximus* muscle, and then vertically downwards along the lateral border of the *biceps femoris*. In this way, after full reflexion of the lower border of gluteus outwards, the entire course of the sciatic nerve from its origin to its passage into the thigh was exposed, and suture of it was relatively easy.

ARTHUR E. BROWN.

Reviews.

A Handbook of Allergy for Students and Practitioners. By WYNDHAM B. BLANTON, M.A., M.D., Litt.D.; 1942. Springfield, Illinois: Charles C. Thomas. London: Baillière, Tindall and Cox. 5¼″ × 9″, pp. 190, with 20 illustrations. Price: $3.00.

WYNDHAM B. BLANTON'S "Handbook of Allergy for Students and Practitioners" is an excellent contribution to the subject. It is divided into three parts. The first is concerned with the fundamentals of allergy and includes a brief discussion of its ætiology, with a statement on methods of performing skin tests and on some principles of treatment.

In Part II the causes of allergy are discussed at length under the headings of inhalants, ingestants, absorbents, injectants and contactants. In discussing pollen allergy the writer stresses the importance of ragweed as a potent factor in the United States of America. It is of interest to note that in Australia this pollen is practically non-existent. The author refers to the great difference of opinion concerning the clinical significance of fungus allergy. Van Leeuwin in Holland regarded it as of the greatest importance in that country. It probably plays a very minor rôle in Australia. In discussing perennial allergy, the author emphasizes the frequency of allergic reactions to orris root, house dust, animal emanations and kapok.

Part III is entitled "The Results of Allergy", and is concerned with allergy of the nose, asthma, dermatological allergy and gastro-intestinal allergies. Preseasonal, coseasonal and perennial methods of treatment of hay fever are explained in detail. In the perennial method of treatment a warning is issued concerning the care which is necessary in changing from an old dilution to a new dilution of an extract and the methods of safeguarding against general reaction are indicated. In the treatment of asthma no reference is made to the use of acetylsalicylic acid. It has been found by some workers that by itself or in combination with ephedrine and phenobarbital, with which it seems to have a synergistic effect, acetylsalicylic acid is of the greatest therapeutic value in a large proportion of cases. Naturally this statement does not apply to the small percentage of people who are sensitive to aspirin.

The illustrations add to the value of the book. Advice is given concerning the preparation and maintenance of a dust-free room. This excellent book concludes with a discussion of various diets suitable for allergic states.

Ear, Nose and Throat Nursing. By JAS. HARDIE NEIL, D.S.O., C. de G., F.R.A.C.S., F.A.C.S.; Third Edition, 1942. New Zealand, Auckland: Clark and Matheson Limited. 8½″ × 5″, pp. 126.

THE excellence of this book is belied by its modest title and format. It contains a wealth of technical information far beyond the needs of the most skilled nurse; coupled with this is mature clinical observation and wisdom, the product of a rich and varied experience. There could hardly be a more useful guide for the house surgeon coming to the ear, nose and throat unit after having had adequate experience in surgical and medical wards. It should prove invaluable to the general practitioner who wishes to acquire particular skill and knowledge of the essential principles of nose and throat work. The illustrations and line drawings cannot be too highly praised and their reproduction is excellent. Our only regret is that, had the book been planned and written from a rather different standpoint, it could have been the best textbook yet available for the medical student; even as it is it is practical and not overloaded with detail.

Surgical Care: A Handbook of Pre- and Post-Operative Treatment. By R. W. RAVEN, F.R.C.S.; 1942. London: Edward Arnold and Company. 4¾″ × 7¼″, pp. 279, with 80 illustrations. Price: 10s. 6d. net.

THAT two booklets on after-treatment and surgical care should be published in the same year on the routine practice of two of the leading London hospitals is an indication that in the surgery of today pre-operative preparation and careful post-operative "care", not only in the wards but in the out-patient "follow-up" departments, are, with modern standardized technique, of almost as great importance as the actual operation itself. Mr. Raven has produced an extremely concise and useful little book based on the surgical ward work at Saint Bartholomew's Hospital. In it he deals briefly with general surgical

principles and pathology in his earlier chapters, touches concisely on the subjects of anæsthesia, radiotherapy and physiotherapy, and then proceeds to describe in detail and without superfluity the routine pre-operative and post-operative treatment of all the usual surgical operations, including also the surgical care of ophthalmological, neurological, gynæcological and orthopædic conditions. The two chapters on urinary surgery in particular are most complete and up to date. Chapters on bandaging, oxygen therapy, blood transfusion, clinical laboratory investigations and radiological examinations are also included.

The subject-matter is very up to date, and this little volume, of convenient pocket size, might well be described as an ideal *vade mecum* for the newly appointed resident medical officer, senior student or nurse working in surgical wards. Some excellent illustrations are included, and the work can be thoroughly recommended as a useful guide to those engaged in the pre-operative and post-operative care of surgical patients.

Proceedings of the Royal Australasian College of Surgeons.

COUNCIL MEETING.

THE following matters arising out of the meeting of the Council held on Saturday, April 17, 1943, are published for the information of Fellows.

Election of Council.

Six nominations were received for six vacancies on the Council. All the retiring members of the Council were renominated and the following were declared reelected: F. Gordon Bell, Sir Hugh Devine, H. R. Dew, Sir Alan Newton, H. R. G. Poate and Sir Robert Wade.

Election of Office-Bearers.

The following appointments were made:

President: Sir Alan Newton.
Vice-Presidents: H. R. G. Poate, F. Gordon Bell.
Censor-in-Chief: W. A. Hailes.
Honorary Treasurer: Balcombe Quick.

Appointment of State and Dominion Committees.

The following appointments were made:

New South Wales: V. M. Coppleson, B. T. Edye, E. M. Fisher (as deputy for I. Douglas Miller), T. M. Furber (as deputy for F. Brown Craig), J. W. S. Laidley, T. W. Lipscomb (as deputy for A. M. McIntosh), J. C. Storey (as deputy for A. J. Aspinall).

Queensland: Hedley J. Brown, A. E. Lee, H. S. McLelland, J. J. Power, A. D. D. Pye (as deputy for J. C. Hemsley), Neville G. Sutton.

South Australia: R. M. Glynn, I. B. Jose, L. C. E. Lindon, R. E. Magarey (as deputy for B. H. Swift), P. S. Messent, L. A. Wilson.

Tasmania: F. W. Fay, B. Hiller (as deputy for J. Bruce Hamilton), H. W. Sweetnam (as deputy for D. W. L. Parker).

Victoria: A. Fay Maclure, J. Newman Morris, W. A. Hailes, Henry Searby, C. Gordon Shaw, John H. Shaw, B. T. Zwar.

Western Australia: J. P. Ainslie (as deputy for F. J. Clark), H. B. Gill, D. D. Paton.

New Zealand: Frank Macky, D. S. Wylie, David Whyte, P. Stanley Foster, J. Leslie Will, J. A. Jenkins.

Admission of New Fellows.

The following Fellows were admitted by the Council:

New Zealand: General Surgery—John Cairney, Desmond Patrick O'Brien, Richard Orgias.

Gift from the Royal College of Surgeons of England.

At the Council meeting, J. Newman Morris delivered to members the first edition of Willis's "Anatomy", a gift from the President and Council of the Royal College of Surgeons of England to the Royal Australasian College of Surgeons. Every member of

the Council was touched and gratified by the gift, which will be preserved with care and with pride, and which adds yet another to many gracious and generous actions which have been taken by the English College to help the Australasian College.

The College is particularly glad to possess a book illustrated by Sir Christopher Wren. Its Fellows share with their English colleagues feelings of wrath and sorrow for the wanton destruction of so much of Wren's work and all are eager to help repair the ravages of war which have been suffered in the homeland, particularly those sustained by the English College.

The President has forwarded a message of cordial greetings and thanks from the Council of the Royal Australasian College of Surgeons to the President and Council of the Royal College of Surgeons of England.

National Health and Medical Service.

The Council adopted the following report:

Essential Principles in any National Medical Service in which a Sound Surgical and Surgical Specialist Service is to be Included.

1. The full benefits of the most efficient medical and surgical treatment must be available to every member of the community.

2. That in any reorganization of medical practice as full a measure as possible of free choice of family doctor or specialist should be preserved.

3. Control of a national medical service must be vested in a body in which all sections of the medical services are adequately represented, and should be divorced from governmental and political control.

4. In any national medical service there should be a cash medical benefit for specialist service which could be accepted in lieu of the specialist service available under the scheme. The object of such a cash benefit would be to enable the patient to exercise his right of a free choice of medical attendant or specialist. The cash benefit would thus permit him, if he did not wish to be treated by the medical personnel provided by the national medical service, to obtain treatment either from other medical men in this service or from doctors in private practice.

5. In a whole-time salaried medical service to be applied throughout Australia, as set out by the National Health and Medical Research Council, it is our opinion that this freedom of choice would not readily be possible. We are also of the opinion that a more effective service could be provided by a process of evolution on the basis of the present system.

6. This would not exclude the possibility of whole-time salaried officers being provided in remote areas.

Essential Principles in Specialist Surgical Service.

1. Surgeons and surgeon specialists must possess approved surgical qualifications based on training, academic qualifications and experience, and granted by a recognized professional body.

2. Surgical and surgical specialist services should be staffed only by such properly qualified surgeons and surgeon specialists. The services of these specialists should be available to all members of the community, even to those in the most remote parts. This would necessitate an adequate supply of properly trained surgeons and specialists. It would also require that special financial arrangements would have to be made to provide surgeons and surgeon specialists in the sparsely populated districts. The provision of a sufficient number of adequately trained surgeons and surgeon specialists would require an organized scheme for the post-graduate training of this personnel.

3. All public hospitals should be expanded into community hospital systems. Hospital systems on this basis should, as the demand arises, be infiltrated throughout Australia.

4. The services of surgeons and surgeon specialists in these community hospital systems should, as far as possible, be part time, so that the rich experience gained by them would be available to all sections of the community. These services should be on a paid basis in teaching hospitals, and surgeons and surgeon specialists would be responsible for undergraduate and post-graduate training.